ARCHIVES OF OPHTHALMOLOGY.

RECURRENT HEMORRHAGES IN THE RETINA AND VITREOUS FOLLOWED BY RETINITIS PROLIFERANS IN BOTH EYES IN A YOUNG MAN WITH SURGICAL TUBERCULOSIS.[1]

BY DR. CHARLES J. KIPP, NEWARK, N. J.

(With two drawings on Text-Plates XX.–XXI.)

W. C., eighteen years of age, consulted me in 1902 for convergent strabismus of right eye. Family history: His father died some years ago of tumor of the brain; particulars are not known. His mother is living and in good health. He has a half brother and a half sister, both the children of his father; they are both in good health. He does not remember to have had any of the ordinary diseases of childhood. He has had attacks of earache many times and his right ear has been and is now discharging pus. He was treated for some eye disease when six years old. He has had much pain in left leg and hip for many years and on August 1, 1901, he was operated on for hip-joint disease by Dr. Jos. A. Blake, who in a letter to me says that he found a small tuberculous abscess in the hip joint, with absorption of most of the head and neck of the femur. The joint was nearly ankylosed at 90° flexion. The remnant of the head was removed and the diseased tissues cut out. He has no report of an examination for tubercle bacilli, but he has no doubt that the process was tuberculous. He recovered from the operation and has been in fair health since.

The examination of his eyes revealed a paralysis of the external rectus muscle of the right eye, for which I could find no cause, although a careful examination was made

[1] Read before the Section on Ophthalmology of the New York Academy of Medicine, March 18, 1909.

of his kidneys and of his nervous system. In the left eye there were several maculæ in the nasal half of the cornea, otherwise both eyes were normal in every respect. Both eyes, V = $\frac{6}{5}$

The examination of his left ear showed a chronic suppuration of the middle ear with caries of the tympanic cavity. I prescribed iodide of potassium in small doses and advised him to live out of doors as much as possible and to eat plenty of good nourishing food.

I did not see him again till October 14, 1905, three years after his first visit. He came to consult me with regard to great impairment of vision of both eyes. He told me that the convergent squint had disappeared in the course of a year after his first visit.

A year ago his left eye was struck by a piece of wood; he had much pain, but did not notice any impairment of sight till recently. I found that the left eye was without signs of inflammation, there were several maculæ in both inner and outer portions of the cornea, and that the iris was torn from its ciliary margin at the temporal side, causing a triangular opening in the iris. The pupil dilated ad maximum after instillation of a solution of atropin. The iris was not discolored. The lens was clear. The vitreous body was so full of dense floating opacities that no details of fundus could be made out. V reduced to movements of the hand.

The right eye was normal in appearance. The paralysis of the external rectus muscle had entirely disappeared. The ophthalmoscope showed cloudy vitreous. The optic disk is normal in appearance. The retinal veins are full and tortuous and there are extravasations of blood, mostly round in form, in all parts of the retina. V = $\frac{6}{10}$; V. F. intact. Tn.

Three weeks later I noted in *right eye:* vitreous very cloudy; many small blood clots in the vitreous. The retina is now in parts opaque, in other parts it is hidden by a thin membranous formation in front of it, and on these membranes there are numerous small vessels of light red color. Some of the membranes are almost transparent. In some places a vessel of considerable size (about the size of a primary branch of the central retinal artery) is seen to come through the membranes and a short distance from its entrance is broken up in numerous very small light colored branches. Some of these grayish membranes come some distance into the vitreous. The extravasations in the retina are still there, some of a bright red, others of a dark red color. The outline of the optic disk is blurred. The retinal veins are very full and tortuous. V = $\frac{6}{15}$.

To Illustrate Dr. Kipp's Article on "Recurrent Hemorrhages in the Retina and Vitreous Followed by Retinitis Proliferans in Both Eyes in a Young Man with Surgical Tuberculosis

Dr. A. Braun, pinxit. Left Eye

Left eye.—The vitreous is much clearer; clumps of blood are now distinctly made out among the floating opacities in the vitreous. The disk is visible. Its outline is blurred. The retinal veins are very full. There are numerous hemorrhages in all parts of the retina. In some parts the retina is covered with thin grayish membranes on which many sharply defined, light colored vessels are seen. V = $\frac{6}{60}$; V. F. intact.

An examination of his left ear revealed a large polypus, the end of which protruded from the external meatus.

November 13th.—He was admitted to the Newark Eye and Ear Infirmary. The *right eye* at this time was as follows: The membranous formation in the vitreous is now of a decidedly grayish color and has now advanced forward. Its surface is best seen with a +8 D glass. The membranes are fixed; they do not change position with the movements of the globe. They are not all on same level. They are all covered with numerous light colored vessels. The fundus is visible only at the periphery.

The *left eye* has improved. The vitreous still contains many small clots of blood, but on the whole the vitreous is much clearer. The retinal extravasations are being absorbed. The patient was shown at the November meeting of the N. Y. Ophthalmological Society and the ophthalmoscopic drawings (Text-Plates XX.–XXI.) were made by Dr. Braun at the same time. An examination of the blood was made at this time and found entirely negative. The urine was free from albumin and sugar. He had no cough and was apparently free from other diseases.

November 18th.—Radical mastoid operation on the left side. The operation revealed vast destruction of the bone. Cholesteatomatous masses were found in the mastoid antrum and in the tympanic cavity. Microscopic examination of the bone and granulations removed failed to discover tubercle bacilli.

During the month following the ear operation his temperature was taken every four hours. It varied from 99.5° to 100°. Later on he had an attack of tonsillitis, during which the temperature rose to 103°. Afterward there was not more than 0.5° difference between morning and evening temperature. During the month of December the right eye remained in about the same condition. The membranous formation in the vitreous gradually advanced somewhat forward, so that its surface could be best seen with a +13 D glass. The opacity of the formation had increased so that a red reflex could be obtained only from the temporal periphery. Here small clots of blood were seen.

The *left eye* gradually became clearer. The clots were absorbed and the fundus was seen pretty distinctly.

During the following five months, the eyes remained in about the condition last described.

In July the examination of the *right eye* showed that the membranous formation had become more opaque, it was in parts almost white, it seemed to fill the vitreous chamber from side to side, leaving only a space on the temporal side free from it. It had apparently also advanced still more. The most notable change was, however, in the number of vessels previously seen on the formation. They had greatly diminished in number and were in some parts of a much darker color than formerly.

On July 16th I noticed for the first time, while the pupil was widely dilated from atropin, that I could see the grayish white formation with the vessels in it, while he was facing a window in daylight. Looked at in this way the formation seemed to tremble with the movements of the globe, but the examination with oblique illumination failed to confirm this. The formation examined by oblique illumination extended most forward on the nasal side, where it seemed to approach the ora serrata; outward, upward, and downward it did not come forward as far; in the visual line it came least forward. Here it was best seen with +15 D behind the mirror. On its lower portion the vessels seemed to be of a darker color than they were elsewhere. On the temporal side, the formation had a sharply defined margin, outside of this a red reflex was obtained in which small dark red clumps were seen. He could not see movements of the hand with this eye.

The *left eye* was pretty free from opacities in the vitreous, otherwise there was no change in it. With 1 +D cyl. 180° V = $\frac{1}{5}$. His general condition is good. His height is 5 feet, 6½ inches; his weight, 147.25 pounds.

August 2d.—Right eye, very little change. There are now several densely opaque stripes radiating from centre of mass, up, in, and down.

The *left eye* which up to yesterday was steadily improving in sight is now again much worse. The vitreous is again so cloudy that details of fundus can scarcely be made out. He knows of no reason for this sudden deterioration of vision unless it is due to pretty severe use of his eye. He has been reading a good deal in the last week, contrary to my advice. His general health is about the same as it has been for several weeks.

August 4th.—Left eye. The opacities in the vitreous are increased. Among the grayish membranes in the lower part are white flakes. There are now also

numerous new extravasations in different parts of the retina.

The *right eye* is unchanged.

April 7, 1907.—The left auricle and surrounding skin are of a bright red color and are much swollen; a blister on the free edge. Temp. 101.5°.

April 8th.—Has now well marked erysipelas of auricle and left cheek. Temp. 104°. Was transferred to General Hospital from which he returned to the Eye and Ear Infirmary two weeks later in excellent general condition.

On November 15, 1907, before I had heard of any deleterious effects following the instillation of a 1% solution of old tuberculin in the conjunctival sac, I instilled a drop of a ½% solution of old tuberculin in the conjunctival sac of the right (blind) eye. This was followed eight hours later by considerable injection and swelling of the palpebral conjunctiva, and twelve hours after the instillation by a muco-purulent discharge. The conjunctivitis lasted for about thirty-six hours, and then passed away leaving the eye no worse than it was before. During the following year he had several times new extravasations of blood in the retina of the left eye especially at the lower periphery, and also in the vitreous, which, however, had all been absorbed by June, 1908.

Since then there have been no new ones, and at the present, March 15, 1909, the eye is free from floating opacities in the vitreous, the fundus is about as pictured in the drawing made by Dr. Braun minus the hemorrhages in the retina. During the last two years he has had no treatment for his eyes or ears; he is in excellent health and now weighs 167 pounds. The left eye has vision equal to ⅝. His V. F. is of nearly normal dimensions for white and colors, but he has an absolute scotoma of moderate size in the lower temporal part of the field.

Briefly summarized, the history shows that this tuberculous individual had an inflammation of his eyes while six years of age. The scars now present in the cornea make it probable that this was a phlyctenular keratitis. He had also about this time a purulent inflammation of his middle ears. In his seventeenth year he had a tuberculous disease of his left hip-joint with absorption of most of the head and neck of the femur. In his eighteenth year he had a paresis of the external rectus muscle of the right eye from some unascertained cause; this passed away in the course of a year. In his twentieth year the left eye was struck by a piece of wood,

causing an iridodialysis at the temporal side, and probably also a circumscribed chorio-retinitis, of which the scar is still visible. A year later the vitreous of this eye was so full of floating opacities that the fundus could not be seen. In his twenty-first year he had numerous hemorrhages in the retina and vitreous in the right eye, which had not sustained a traumatic injury. This was followed by a retinitis proliferans which destroyed the vision of this eye. In the left eye the hemorrhages in the retina and vitreous were also followed by retinitis proliferans, but of much less extent, and by many more attacks of hemorrhage in the retina and vitreous during the following two years. All of these extravasations were, however, absorbed without having produced additional visible changes in the retina or vitreous. In the same year the chronic purulent inflammation of the left middle ear gave rise to grave symptoms, and a radical mastoid operation was made which revealed vast destruction of the temporal bone. A year and a half later there was an attack of erysipelas of the external ear of the operated side. In his twenty-fourth year, the Wolf-Eisner Calmette conjunctival test with ½% solution of old tuberculin produced a well-marked reaction.

In his twenty-fifth year he is in excellent health. The *right eye* is blind from retinitis proliferans, while the vision of the *left eye* is equal to $\frac{6}{9}$ and the retinitis proliferans seems to be arrested.

Whether or not in this case the eye disease was of a tuberculous nature, must remain undecided. The recurrent hemorrhages in the retina and vitreous of the left eye might have been due to the traumatic injury received a year before I noticed them. But the recurrent hemorrhages and the subsequent development of the proliferating retinitis in the right eye could not be ascribed to such a cause. Tuberculous disease of the retinal blood-vessels is spoken of as the cause of the recurrent hemorrhages, and such a condition of the vessels may have been present in my case. If such was the case recovery from this condition must be possible, for no hemorrhages have occurred in the left eye of my patient during the past year. It seems useless to speculate further with regard to the pathology of this case.

In connection with this case allow me to call your attention

to a case of hemorrhage in the vitreous which I have reported in the *Transactions of the American Ophthalmological Society* for 1895. In this case a boy ten years of age, when first seen by me, was blind in the right eye. The vitreous was so cloudy that the fundus could not be seen. No blood clots could be seen in the vitreous. Afterward a whitish membrane with vessels on it developed behind the lens and later a cataract obstructed the view. Fifteen months later he had a profuse hemorrhage in the vitreous of his left eye which blinded him for a time. A year after this attack vision was equal to $\frac{1}{5}$. Six years after this first attack the left had another attack of hemorrhage in the vitreous, which again blinded him for many months. Six months after the beginning of his second attack his vision was equal to $\frac{1}{5}$ with +0.75, c. 90°. During the last fifteen years his eyes have remained in good condition. When last seen a year ago he complained of muscæ volitantes. The eye was apparently normal except a few small spots of atrophy in the periphery of the choroid and a few very thin almost transparent membranes in the periphery of the vitreous; with +0.75, c. 90°, V = $\frac{1}{1}$. He is now in excellent health. When I first saw him he was also in good general health, but afterwards he lost flesh and had a cough for which his physician directed him to live out of doors. He has thus far shown no symptoms of tuberculosis of the lungs or any other part. His father, whom I knew very well, was suffering from tuberculosis of the lungs when he first brought his son to me, and he died from the disease some years later. The boy's mother is living and in good health and so are all of his brothers and sisters. I have brought this case again to your notice, as it may possibly have been one of incipient tuberculosis, terminating in recovery.

I have also seen a number of cases of hemorrhage in the retina and the vitreous in young men suspected of having incipient tuberculosis of the lungs, but I shall not weary you with a detailed history of them, as no tubercle bacilli were found in their sputa and their lung disease seems to have been cured. The blood was absorbed and vision restored in all of these cases.

On the other hand I have seen in a number of cases in

young people in perfect health and without the least sign of a tuberculous focus anywhere, hemorrhages in the retina and vitreous at intervals of time varying from a year to six years, and I have also seen retinitis proliferans follow extravasations of blood in the retina and vitreous in persons old and young in whom no signs of a tuberculous focus could be discovered at the time the eye disease made its appearance, nor in the following years.

In the latest publication on this subject, Wilbrand und Saenger, *Die Neurologie des Auges*, IV Band, Erste Haelfte, 1909, p. 218, I find the following: The etiology of the recurrent juvenile extravasations of blood in the retina and vitreous is still shrouded in obscurity. The correspondence of the cases as to the disease picture and the course of the affection, characterized by its occurrence preferably between fifteen and twenty-five years of age, by the profuseness of the extravasations, by the relatively speedy resorption of the blood and by a marked tendency to recurrence, make it probable that the cause of the disease is the same in all. In general, we can divide the cases into two main groups, one comprising the cases of congenital, and the other of cases of acquired friability or rather permeability of the walls of the blood-vessels, and in such cases occasionally agitation or stasis of the blood current will cause the occurrence of the extravasations. Among the cases cited as illustrations of the acquired group, is given a case reported by Rosenfeld in which tuberculosis of the lungs existed at the time the recurrent extravasations of blood in the vitreous occurred. This reporter in this connection reminds us that von Michel in his text-book has stated that extravasations of blood in the vitreous are sometimes the first sign of tuberculosis of the ciliary body.

A somewhat extended search through the literature of this subject has failed to discover the record of a case like the one here reported in detail.

TO ILLUSTRATE DR. CZERMAK'S ARTICLE ON "A FIBROUS PSEUDO-LENS TAKING ITS ORIGIN FROM THE TUNICA VASCULOSA LENTIS".

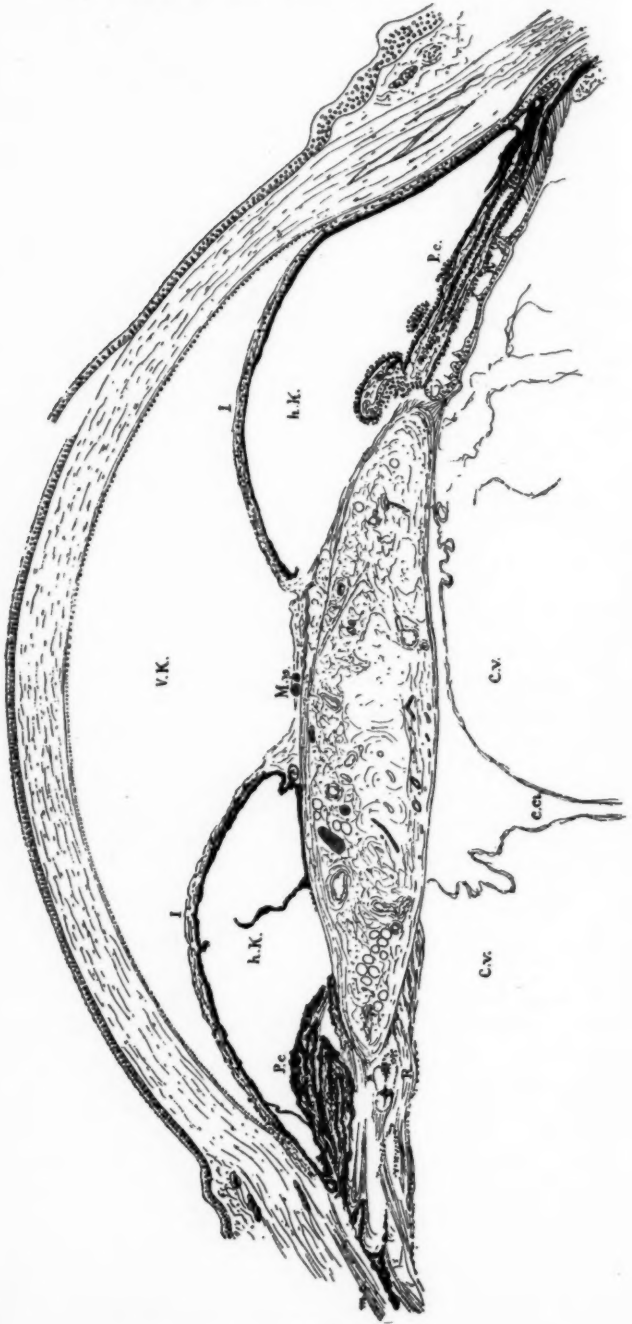

FIG. 1.

A FIBROUS PSEUDO-LENS TAKING ITS ORIGIN FROM THE TUNICA VASCULOSA LENTIS.

By the late Prof. WILLIAM CZERMAK, Prague.
Presented and Completed by Dr. ULBRICH.

(*With six figures on Text-Plates XXII.–XXV.*)

Translated by Dr. Alfred Braun, New York.

THE anatomical preparations of the following case were presented by Prof. Czermak at the second session of the meeting of the German Ophthalmological Society, in 1905.

CLINICAL HISTORY.

Joseph P., a laborer, twenty-five years of age, appeared at the clinic, on Dec. 15, 1904.

At the age of three months, he had had a rash, coincident with an inflammation in his left eye. He does not remember ever to have seen with this eye. Ten years ago, he had pain in the left temple, which lasted several days. Six weeks ago, his left eye became inflamed and very painful. His physician made a diagnosis of corneal inflammation, and prescribed eye-drops at first, and then an ointment. The inflammation increased. He was sent to the clinic by another physician.

OBJECTIVE EXAMINATION

Surroundings of the eye, and lachrymal apparatus: normal.

O.D.: Horizontal nystagmus, pupil slightly irregular in shape, hyperopia of 4 to 5 D. Ophthalmometer shows 2 D of astigmatism. Vision = 0.5. O.S.: Photophobia and lachrymation. Conjunctiva congested, with pupillary hypertrophies. The eyeball, diminished to half its size, is in position of adduction. Mobility, outward, is limited.

Horizontal nystagmus. Ciliary injection. Near the centre of the cornea, a few superficial ulcerations, which seem to have been the result of the rupture of vesicles. The anterior chamber is deeper in the middle than at the periphery, where there is a bulging forward of the iris. The iris is atrophic and very vascular. The pupil, *3mm* in diameter, is immobile. The iris forms a funnel, at the bottom of which lies the pupil, which is filled in by a yellowish-white membrane. Tension is somewhat increased. Amaurosis.

December 18th.—The corneal ulcer was covered by a yellowish deposit.

December 20th.—The eyeball was enucleated under chloroform anæsthesia.

December 23d.—Patient was discharged.

DESCRIPTION OF THE SPECIMEN.

The eye was fixed in formol, and hardened in alcohol. It had an axial diameter of *22mm*, and a frontal diameter of *19mm*. After hardening, the upper portion of the ball was removed by making a horizontal incision, *3mm* above the corneal margin. The lower portion was embedded in celloidin, and a portion divided into horizontal sections. The remainder of this portion and the upper portion were divided into vertical sections. The sections were stained with hæmalum-eosin, with Van Gieson's stain, and with Russel's stain.

After division of the eye into an upper and lower part, it was seen that the choroid and retina were in proper position, and that the vitreous, which filled out the entire interior, was filled with a whitish mass, which had probably coagulated as a result of the hardening process.

If a frontal section, which passes through the centre of the pupil, is examined with the naked eye, there is seen the ordinary picture of a bulging iris, attached at its pupillary border to the capsule of the lens. The ciliary processes are long-drawn out, and attached to the border of the lens. The lens itself seems to be considerably flattened, shortened in its frontal diameter, and spindle-shaped.

This lenticular mass has a frontal diameter of *6mm*, and a sagittal diameter of *1.25mm*. On meridional section, it has a form which reminds one of the alga "pleurosigma angulatum," the familiar test-object. This mass is held in position by the following connections (Fig. 1):

1. It is attached at the middle of the anterior surface to the pupillary margin of the iris.

2. The ends of the elongated finger-like processes of the ciliary body are attached to the outer part of its an-

To Illustrate Dr. Czermak's Article on "A Fibrous Pseudo-Lens Taking its Origin from the Tunica Vasculosa Lentis"

Fig. 2.

U of M

terior surface. Its unpigmented epithelium is attached to the anterior surface of the lenticular mass, and the zonular fibres, which lie between and behind the ciliary processes, run into the superficial layers of the "lens," on the anterior surface, and into the edge.

3. Behind the ciliary processes, at the edge of the mass, and to a portion of its posterior surface, is attached the anterior border of the retina, the limitans of the retina being adherent to the most superficial layers of the "lens."

There exists in this way (except for a small space at the lateral margin, where the retina does not quite reach to the border of the lens), between the posterior surface of the ciliary processes and the anterior surface of the retina, a triangular space, with the apex directed outward, in which the edge of this mass lies, like a lens in its frame (Fig. 2).

The vitreous lies close to the posterior surface of the mass in places, but with a distinct line of separation, while in other parts a fine cleft is seen. At the periphery, the connection is closer.

In the region where the border of the retina does not reach the edge of the "lens," the space, up to the posterior surface of the ciliary processes, is filled in with vitreous, in which run the equatorial and posterior zonular fibres. We have here, therefore, the same conditions as in the embryo.

The lenticular mass consists of a firm connective-tissue cortex, and a nucleus, which is composed of a tangle of looser and firmer connective-tissue fibres and vessels.

The cortex consists of connective-tissue fibres, which run parallel to the surface, and are seen running partly meridionally and partly in cross-section. The anterior cortex, which is much thicker than the posterior, contains a variable number of spindle-shaped nuclei and a moderate number of blood-vessels. All the vessels of the cortex are continuous with those of the nucleus. The anterior cortex also contains remains of the lens capsule. In places, two layers can be demonstrated: an anterior, which is covered by a thin layer of connective tissue, or lies directly on the surface of the lenticular mass, in which case it is adherent to the iris; and a deeper layer, which lies in the posterior portion of the anterior cortex. In some sections, in the region of the equator, the anterior capsule may be seen to pass into the posterior layer in the form of a loop. Whether this posterior layer is the posterior capsule, cannot be determined. At all events, these remains of the capsule are very insignificant, and are only

seen in portions of the central region of the anterior surface.

At one point of the posterior surface of the lenticular mass, the cortex contains no nuclei, is homogeneous, and lighter than the rest, so that one might suspect a portion of the posterior capsule. But even disregarding the fact that this tissue is seen at its margins to be continuous with the surrounding fibrillary cellular tissue, it is seen in eosin preparations to be more fibrous than capsular tissue, and less light and glassy. With Russel and Van Gieson stains, it is seen that this tissue has nothing to do with the posterior capsule, but is probably vitreous.

The vessels which run through the cortex arise from the nucleus. They bend at the margin of the latter and then run parallel to the surface. They go to the outermost edge of the mass, between the ciliary processes and retina, but no vessel passes from the cortex to either of these structures. On the posterior surface, a few small vessels pass into the vitreous, at the margin of a funnel-shaped defect of the vitreous, which is opposite the posterior pole of the "lens." The base of this funnel-shaped defect lies on the posterior surface of the "lens," and its apex is directed backward (canal of Cloquet).

The nucleus of the mass consists of a spongy tissue, which, in places, looks not unlike normal iris tissue, without pigment (Fig. 3). Its network is denser and more elongated near the cortex. It consists of very delicate fibres, and contains many spindle-cells and three-rayed cells. There are also regions consisting of dense wavy connective-tissue fibres, with few cells. This dense tissue is seen especially in the central portion of the nucleus, and is connected with the adventitial sheaths of the larger vessels.

We find here many vessels, of which the largest has a lumen of 60μ. They show a well developed wall, with an adventitia of greater or less thickness. The arteries show a circular muscular sheath of three layers, which closely resemble the thick arteries of the iris. One large vein shows a dense round-celled infiltration of its walls. Many vessels show sclerosis of their walls. In some of the smallest vessels, the lumen is entirely obliterated, through enlargement of the endothelial cells.

In some portions of the nucleus, especially in the central portions, there are many large round vacuoles, about 56μ in diameter, which look like empty fat cells. As the preparation was heated with ether-alcohol, this point could not be definitely determined.

The arteries arise from the larger arteries of the iris, which pass through the pupillary margin into the "lens."

To Illustrate Dr. Czermak's Article on "A Fibrous Pseudo-Lens Taking its Origin from the Tunica Vasculosa Lentis"

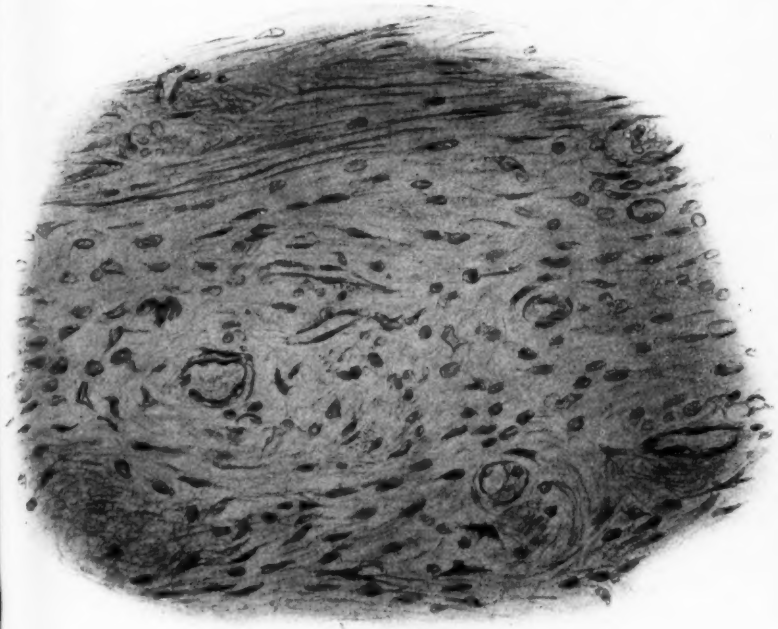

Fig. 3.

Fig. 6.

In the same manner, the veins of the "lens" pass into the pupillary margin of the iris.

The iris is very thin, its tissue very dense, and the sphincter muscle well preserved. It is bulged forward, and its entire periphery is adherent to the posterior surface of the cornea, thus closing off the spaces of Fontana. The pupillary portion extends back in a funnel shape, so that the pupillary margin is at right angles to the surface of the lenticular mass. A thin membrane, which shows the structure of iris-tissue, fills in the pupil.

The insertion of the iris into the "lens," on the lateral side, is very thin at one point, at which the pigment layer is pent around in a spur-formation, and lifted away from the cortex. In the space thus created, there is a piece of lens capsule, which can be followed from the point of insertion of the iris almost to the margin of the lenticular mass. From the insertion of the iris, a process is sent over the anterior surface of this piece of capsule, which, becoming gradually thinner, finally appears as a thin perforated membrane, which contains many pigmented, branched cells.

The ciliary processes are thinned, and full of dilated vessels. They are elongated, and look not unlike a papilloma on section. The pigmented and the unpigmented epithelium is normal.

Between the ciliary processes, to a slight extent, on its anterior surface, and in the space between its posterior surface and the prolongation of the retina, are seen normal bundles of zonula fibres. The fibres reach the anterior surface of the pseudo-lens, except in that region where the retina does not reach the pseudo-lens, where the fibres reach the edge and posterior surface of the pseudo-lens.

The dilated posterior chamber contains septa composed of pigmented epithelium, which stretch from the posterior surface of the iris to the anterior surface of the pseudo-lens, or ciliary body. The septa are not complete.

The posterior surface of the hindmost ciliary process is covered by non-pigmented epithelium, whose high cylindrical cells resemble those of the normal orbiculus ciliaris. This can be explained on the assumption that, as a result of traction, the point of origin of the ciliary processes was raised in a fold. This seems to be opposed by the fact that the ciliary muscle passes smoothly over the entire region of origin of the ciliary processes, and is not also drawn up into a fold (Fig. 4). It passes backward a normal distance from the ciliary processes.

The retina covers the inner surface of the ciliary body at least to the point of origin of the last ciliary process,

to be changed there into the pars ciliaris retinæ (if we take the region where the posterior end of the ciliary muscle lies as the position of the ora serrata). Everywhere, except in the lateral region, the retina passes from this point parallel to the ciliary processes, to the edge of the pseudo-lens, and to the posterior surface, to which it is attached for a short distance. In some regions, it reaches to the junction of the outer with the middle third of the posterior surface. The membrana limitans of the retina is firmly attached to the cortex of the pseudo-lens and follows all the irregularities of its surface.

The retina is attached to the choroid, except for a small distance at the ora serrata, where there is a slight detachment. The posterior portion of the retina is normal, except that the nerve-fibre layer and ganglion-cell layer are missing. In the region of the posterior end of the ciliary muscle, it takes on a gliomatous character, and from the point of separation from the ciliary body to near its insertion into the pseudo-lens, it is full of numerous small and a few large cystic cavities. Near its insertion, its structure is again normal. It shows here thickened Müller's fibres, an indication of the nuclear layers, and vessels which seem to have some connection with the vessels in the vitreous, behind the pseudo-lens. This portion of the retina is covered on its anterior surface by an epithelial layer, which consists of a single layer in some places and several layers in others, and which seems to be directly connected with the unpigmented epithelium on the last ciliary process, near its root. At the equator of the pseudo-lens, the central end of this layer becomes thickened and branched so that it forms a network. This epithelial network reminds one of a rapidly growing epithelioma (Fig. 5).

In that region where the retina stops at the posterior end of the ciliary processes and becomes continuous with their posterior epithelial lining, a membrane consisting of the unpigmented cells of this layer forms a substitute for the above-described retinal diaphragm. It lies between the anterior and posterior zonular fibres, in the so-called zonular space. Its peripheral origin is varied. Either a cystic cavity separates the epithelium into two layers, of which the anterior remains attached to the ciliary process, while the posterior remains a thin membrane; or a very thin sheet, consisting of two layers, springs from the epithelium of the ciliary process at some distance from the anterior end of the retina. Centrally, the membrane becomes thicker in places, and consists of cylindrical cells. Cavities occur in it, whose walls are lined by flat epithelial cells. Nearer the pseudo-lens, these cavities

TO ILLUSTRATE DR. CZERMAK'S ARTICLE ON "A FIBROUS PSEUDO-LENS TAKING ITS ORIGIN FROM THE TUNICA VASCULOSA LENTIS"

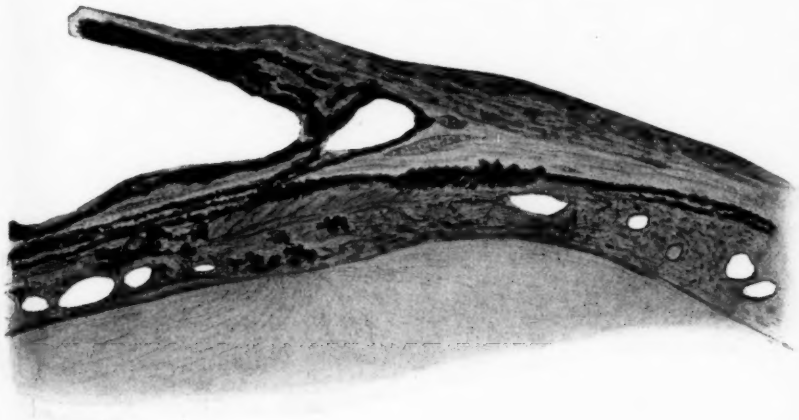

FIG. 4.

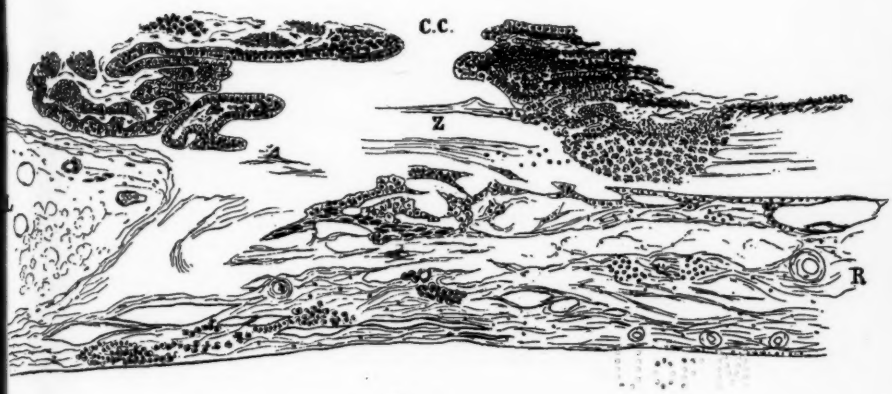

FIG. 5.

become larger, and the partitions and anterior walls become fibrous and show few nuclei, while the anterior ends of the partitions and the posterior walls consist of long epithelial cells. The central end is firm, and consists of large cells, which resemble Müller's fibres, partly, and partly the cylindrical cells of the pars ciliaris retinæ. In places, it consists of two layers of cubical cells.

Thus the entire membrane seems to consist of two layers, of which the posterior becomes continuous with the retina, and the anterior with the orbicular cells. These layers are interrupted in places, through atrophy, sometimes one, sometimes both. In the lower-outer portion of the eye, this membrane ends much farther out in the periphery, so that it consists of a short process which, in places, is difficult to distinguish from the epithelium separated from the ciliary body.

The entire retinal diaphragm gives the impression of a fold, due to the fact that the equator of the pseudo-lens was at one time adherent to the region of the ora serrata, and that with the growth of the ball, and the diminution in size of the pseudo-lens, the retina was drawn centrally into a fold, and torn in places. The portions not attached to the equator remained behind. The posterior layer of the diaphragm seems to consist of retina; and the anterior, of the epithelium of the ciliary processes.

The anterior and posterior chambers are filled with a white coagulated mass and a few round cells.

The vitreous is also full of a white coagulated mass and a few round and spindle cells. Behind the centre of the pseudo-lens is a triangular space, with the apex backward. The choroid is thin and atrophic. The optic entrance is markedly excavated. The sclera is normal.

The cornea is vascular at the margin, and shows increase of nuclei. The bulbar conjunctiva and episcleral tissue show marked infiltration.

In explaining the origin of the pseudo-lens, we may take two viewpoints.

1. We might consider it due to the inflammation concurrent with the skin eruption, which occurred when the patient was three months old.

2. We might consider the condition to be congenital.

A study of the anatomical specimen will show the untenability of the first theory. If we consider the lenticular mass to be the result of the organization of a cyclitic exudation, then we cannot explain the absence of a similar exudate in

the posterior chamber. Equally unexplainable is the absence of vessels passing into the structure from the ciliary processes, the orbiculus ciliaris, or the adherent retina.

We find the lenticular mass entirely separated from the ciliary body and retina, except for the adhesions between its equator and the tips of the ciliary processes and the fold previously described.

The entire posterior chamber—its prezonular portion, which lies in front of the lengthened ciliary processes, its zonular portion, in which the zonular fibres run perfectly free, and its retrozonular portion, which lies behind the retinal fold—contains not the slightest trace of an old organized exudate. The coagulated albuminous mass which it contains is the same as that seen in the anterior chamber and vitreous, is of more recent origin, and has no connection whatever with the origin of the lenticular mass.

The entire vascular system of the mass is in relation only with the iris.

We might consider the mass to be an inflammatory exudate arising from the pupillary margin of the iris. After disappearance of the lens, an exudate might have been poured into the pupil and the lenticular space behind and become organized through vascularization from the iris. But, casting all other objections aside, this theory would be invalidated by the fact that the lens capsule is in contact with the pupillary margin, which under the aforesaid conditions would not be possible. It is also difficult to understand why this exudate should confine itself to the position of the lens, unless it was poured into the lens capsule, which was open in front. But in this case it would have to be entirely surrounded by the capsule, which is not the case.

The entire picture gives one the impression of an organoid change, and not merely of a simple inflammatory product.

I have come to the conclusion that we have to deal with a congenital anomaly, due to a persistence and hypertrophy of a portion (especially the posterior portion) of the tunica vasculosa lentis, with resorption of the enclosed lens, with the exception of a few remnants of the capsule. This theory can explain all the circumstances satisfactorily.

The following speaks for this assumption:

1. The pupil is occluded by a membrane, which shows the same structure as the iris. It is undoubtedly a persistent pupillary membrane.

2. The structure is vascularized entirely by the iris. The arteries in the structure have the same appearance as the arteries of the iris.

3. At the posterior pole, a few vessels pass into the vitreous, which contains here a fine vascular network.

I consider the structure to have been formed as follows:

At a period when the lens capsule and zonula were already formed, there occurred a thickening and hypertrophy of the tunica vasculosa lentis, which had not yet become reabsorbed. What the causes of this hypertrophy were is not known. There might have been inflammatory changes in the uveal tract, which, as often happens in embryonal eyes, healed, and left no other traces of their existence.

Whether the eruption which occurred in the third month of life was of a syphilitic nature, thus making a luetic origin of the intrauterine inflammation probable, cannot be decided, since there is nothing else in the general condition of the patient to suggest lues.

The hypertrophy of the tunica vasculosa lentis led to adhesions with the ciliary processes and margin of the retina on the one hand, and on the other hand (analogous to the observations of Hess in chicken embryos) to rupture of the posterior lens capsule and resorption of the lens. The space resulting from this resorption was filled in by a vascularized growth from the inner surface of the tunica vasculosa. In this way the nucleus of the structure was formed, while the cortex was formed by the tunica vasculosa itself.

With further growth of the eye, as a result of the increase in size of the circle of origin of the ciliary processes and the orbiculus ciliaris, in spite of the fact that the lenticular structure also grew, there occurred a radial traction on the ciliary processes and adherent retina, so that these structures were drawn up into a fold, while the lenticular mass itself, which at the beginning was more rounded, became flattened. At that time, the iris was flat and, as the pigment remains show, adherent in parts to the anterior surface of the mass

by its posterior surface as well as by its pupillary margin. Nevertheless, as in cases of persistent pupillary membrane, the fluid in the posterior chamber probably passed through minute openings in the spongy tissue of the membrane into the anterior chamber.

If absolute seclusio pupillæ and bulging forward of the iris had existed from the beginning, the increased intraocular pressure would have resulted in ectasia of the growing ball, with the formation of congenital hydrophthalmos. On the contrary, we see here a moderate grade of microphthalmos, probably due to interference in growth, as a result of traction of the lenticular mass.

The increased pressure probably came on recently. Pains came on six weeks ago, and increased in intensity, until the eye had to be enucleated. As there were traces of a recent corneal inflammation in the specimen, there probably resulted an iritis, with transudation of an albuminous fluid, which caused a stoppage of the small openings in the pupillary membrane (Fig. 6). The resulting protrusion forward of the iris resulted in contact of its periphery with the posterior surface of the cornea, these adhesions preventing the outflow from the anterior chamber, thus leading to an acute increase of intraocular pressure.

This or a similar explanation would have to be given, if we would explain the condition by a cyclitis or irido-cyclitis at the third month of life, since even at that time there could not be an increase of intraocular pressure without producing an ectatic eye.

We might call the condition congenital aphakia, but it seems to me that in this, as well as in those cases mentioned in the literature where the lens is replaced by some other structure, the presence of this structure dominates the anatomico-clinical picture, and the condition might better be named pseudo-lens, or pseudophakia. We might divide pseudophakia into congenital and acquired, into osseous and fibrous. In my case, we have a congenital fibrous pseudophakia.

Professor Czermak's manuscript, which ends with the collection of literary references given below, was probably only intended as material for a paper to be prepared later.

A study of the literary references given leads to the consideration of some additional points of interest.

Von Grolman's case presents the closest resemblance to the above-described case. He reported a case of microphthalmos without coloboma, with slight bulging forward of the iris, and "cataract" formation. The lens was very white, and a white thread ran from the optic entrance to the lens. There was a funnel-shaped detachment of the retina, and a persistent hyaloid artery. The lens was more spherical than usual, with an irregular surface. The anterior capsule was very thick and irregular, and did not reach quite to the equator. On the posterior surface was a fine fibrillary mass, which was not continuous with the anterior capsule, sent processes to the corpus ciliare and between the adjacent detached retinal folds, and became continuous with the thickened sheath of the hyaloid.

In the interior of the lens, as the only recognizable remains of the lens fibres, were seen those droplets which are observed in every cataract. Where they occurred in larger groups they resembled fatty tissue very closely. To judge from von Grolman's illustration, which agrees fully with the picture of Czermak's case, it was undoubtedly fat. The fat cells were enclosed by delicate connective tissue. There were many vessels, of which the largest entered the posterior pole of the lens. Von Grolman (as Vossius had already shown) erroneously designated the condition as a fibrous degeneration of the lens.

This case is of considerable interest in connection with Czermak's case, inasmuch as it shows that the same histological picture is possible, when two capsule layers are found on the anterior surface of the structure, representing the anterior and posterior capsules of the reabsorbed lens (Vossius); and secondly, that in this case also the structure was not supplied by vessels from the ciliary body or retina, as is always the case when the lens is replaced by connective tissue or bone as a result of inflammation; thirdly, that a vascular supply from the iris is not a necessary postulate to the formation of such a pseudo-lens, as the persistent hyaloid artery may take on this function. (In Czermak's case, also, there were delicate vessels in the vitreous, at the pos-

terior pole, which were continuous with the vessels of the pseudo-lens.)

The shape of the structure, in von Grolman's case, was more spherical. This was due to the fact that the loose union between the retina and choroid, resulting in detachment of the retina, allowed a diminution of the traction which caused a flattening of the structure in Czermak's case. The persistence of the hyaloid artery, which was in contact with the posterior surface of the pseudo-lens, may also have contributed to the preservation of its spherical form.

Thus we do not doubt that von Grolman's case was identical with Czermak's. The variations in his case not only do not speak against Czermak's assumption, that the condition is an hypertrophy of the tunica vasculosa lentis, but this view throws the necessary light on von Grolman's case.

Vossius demonstrated some preparations from a case of microphthalmos congenitus at the Heidelberg meeting.

The white, rounded opacity in the pupil is caused by the lens capsule, which is thrown into folds and contains no lens elements. It lies before the iris, in the anterior chamber, shows in its folds hypertrophied epithelium and a few vessels, and is adherent to the cornea and to the pupillary margin of the iris. To the posterior portion of this lens rudiment is attached a mass of connective tissue, rich in nuclei, which passes backward to the papilla and contains a thin vessel (the persistent hyaloid artery). There is a flat detachment of the retina.

According to Vossius, the adhesions between the iris, the lens rudiment, and the cornea were caused by inflammatory changes in the fœtal eye, after the formation of the lenticular vesicle.

Vossius considered von Grolman's case to be analogous to Lange's case, which will be discussed later.

We can see the same characteristics in Vossius's case, as in the two previously described cases. We see here also a resorption of the entire lens, with the exception of the capsule, behind which is a mass of connective tissue nourished by the hyaloid artery and connected to the iris and retina. The diminution in size of the pseudo-lens, through fibrous change, was much greater here. The fatty tissue in the

pseudo-lens, seen in the first two cases, was lacking in Vossius's case. Signs of a fœtal inflammation were present in his case, but not in the other two cases.

Wiegels reported a case of congenital microphthalmos, with fat in the vitreous. The eye was diminished in all its diameters, and the vitreous filled with fatty tissue, traversed by a well developed arteria hyaloidea, and surrounded by a connective-tissue capsule, except for a small area below and behind. Of especial interest is the fact that this capsule is continuous with the iris tissue at the pupillary margin. At the bottom of the ball, behind the ciliary body, it is continuous with the sclera. The lens seems, at the first glance, to be missing. On closer examination, however, we see behind the upper portion of the iris, surrounded on all sides by connective tissue and fat, a faintly stained capsule, twisted into folds.

To a certain extent, Wiegels's case forms a contrast to Vossius's case. In the latter case, there was contraction of the abnormally developed tunica vasculosa, while in the former case there was a fatty hypertrophy of this structure. Wiegels speaks only of an atypical development of the mesodermal tissue which forms the vitreous, and makes no mention whatever of the tunica vasculosa lentis. However, the tunica vasculosa and the above-mentioned tissue are merely different portions of the same structure, which are supplied by the same vessels. I do not doubt that the tissue mentioned by Wiegels formed the greater part of the fatty tissue. However, the anterior portion represents the abnormally developed vascular membrane of the lens. This can be seen from the closure of the pupil by means of the capsule surrounding the fat, and from the similar folded capsular remains of the reabsorbed lens.

Lange's case was very similar to Wiegels's. A microphthalmos, with coloboma of the choroid below, and thickening of the iris and ciliary body, showed only calcified remains of the lens. Behind the pupil lies a conical mass of fat, of 3*mm* diameter (with the base anterior), obtaining its vascular supply through the hyaloid artery. The detached retina, which lies against this fatty mass, shows no defect corresponding to the coloboma in the choroid. There is another

islet of fat in the coloboma, and a third in the sclera, at the optic entrance. Lange considers his case to be due to metaplasia of the vitreous, or rather of the mesodermal process which enters the optic cleft. At the time of the beginning of the metaplasia the secondary optic vesicle was already closed. No mention is made in this case of a pupillary membrane. From this description, there is no reason why we should not place the position of the anomaly in the anterior part of the mesoderm, *i. e.*, the tunica vasculosa lentis.

Hanke's case must still be mentioned. In his case there was a persistent hyaloid artery, with fat in the vitreous, and a process of tissue in front of the iris, communicating with the orbital tissue at the limbus. The condition was too complicated to make comparisons with the previous cases.

To sum up Czermak's case: through hypertrophy of the tunica vasculosa lentis there resulted resorption of the lens. In its place we find a fibrous structure, connected to iris, ciliary body, and retina, but receiving its vascular supply only from the iris and vitreous.

LITERATURE

MANZ. *Archiv f. Ophthal.*, Vol. 26, Part 1, p. 154.
VON GROLMAN. *Ibid.*, Vol. 35, Part 3, p. 187.
VOSSIUS. *Report of the 25th Meeting of the Ophthal. Society*, p. 294.
LANGE. *Archiv f. Ophthal.*, Vol. 44, p. 66.
WIEGELS. *Ibid.*, Vol. 50, p. 368.
HANKE. *Ibid.*, Vol. 57, p. 38.

EXPLANATION OF PLATES

FIG. 1.—General View. vK, anterior chamber, I, Iris, hK, posterior chamber, Mp, membrana pupillaris, Pc, processus ciliaris, Cv, corpus vitreum, CCl, canalis Cloqueti.

FIG. 2.—The pseudo-lens, pph, lies in a groove between ciliary body, pc, and retina, r, like a lens in its frame; i, iris, c, cortex, n, nucleus of the pseudo-lens; z, zonula, passes to ciliary process, and between the last ciliary process and retina.

FIG. 3.—Nucleus and cortex under greater magnification.

FIG. 4.—The ciliary body passes over the base of the drawn-out fold formed by the ciliary body and retina.

FIG. 5.—Insertion of retina into the pseudo-lens. Cc, corpus ciliare, Z, zonula, L, pseudo-lens, R, retina.

FIG. 6.—The exudate formed during the last inflammation plugs the narrow openings in the pupillary membrane, which serve for the escape of the aqueous.

L, pseudo-lens, Mp, membrana pupillaris, I, iris, E, exudate, C, lens capsule.

ON THE OCCURRENCE OF OPSONINS IN THE AQUEOUS HUMOR.[1]

By Dr. ARNOLD KNAPP, New York.

IT has been shown by several investigators that some of the avascular structures in the eye participate in the general processes of immunity; in other words that antibodies produced in the blood pass into nonvascular parts of the eye, especially into the aqueous humor.

Even before this era, it was demonstrated that the second aqueous humor differed from the first in certain essential ingredients, the aqueous becoming richer in albumen. With the publication of Wright's researches on opsonins, another property of the blood serum was demonstrated of undoubted importance for the processes of immunity.

It seemed to me that the properties of the aqueous humor could be studied along these lines, and three years ago I began some experiments to determine the opsonic power of the aqueous humor under certain conditions. These experiments could not be concluded until the past summer, when certain questions were again gone over under the guidance of Dr. Augustus Wadsworth, Associate in Bacteriology, Columbia University, to whom I am greatly indebted for advice and co-operation.

The course of the investigations was as follows: Rabbits were actively immunized with streptococcus and staphylococcus series. They received repeated doses subcutaneously and intravenously of twenty-four-hour bouillon growths boiled for ten minutes, and finally live cultures were injected. The aqueous was aspirated from the anterior chamber of the immunized animals and from normal rabbits. The latter

[1] Demonstration before the Section on Ophthalmology, New York Academy of Medicine, meeting of February, 1909.

rabbits then after fifteen minutes again had their anterior chambers tapped, thus giving the second normal aqueous humor. Normal salt solution was used as a control. The bacterial emulsion was made with a twenty-four-hour growth in bouillon; a uniform solution was prepared and disintegration carefully sought for. The leucocytes were obtained from a dog, and were repeatedly washed to remove the blood serum. The mixtures were then made with capillary tubes according to Wright's latest technique. The tubes were sealed and placed in a water bath at 37° C. for from fifteen to twenty minutes. The contents spread on glass slides were stained for one-half minute with Jenner's solution. The specimens were then examined under the microscope to determine the amount of phagocytosis which had taken place. A count was attempted, but it seemed sufficient for our purposes to get a general idea of the activity of the phagocytosis. This, I think, was fairly obtained by selecting parts of slides for comparison which contained about the same number of leucocytes and extracellular bacteria.

The following reactions were studied and compared:

1.	Streptococcus + salt solution + leucocytes.	Phagocytosis Slight.
2.	Staphylococcus + salt solution + leucocytes.	
3.	Streptococcus + streptococcus aqueous[1] + leucocytes.	Moderate.
4.	Staphylococcus + staphylococcus aqueous[1] + leucocytes.	
5.	Streptococcus + normal aqueous + leucocytes.	Slight.
6.	Staphylococcus + normal aqueous + leucocytes.	
7.	Streptococcus + 2d normal aqueous + leucocytes.	Pronounced.
8.	Staphylococcus + 2d normal aqueous + leucocytes.	
9.	Streptococcus + normal serum + leucocytes.	Moderate.
10.	Staphylococcus + normal serum + leucocytes.	
11.	Streptococcus + streptococcus serum[2] + leucocytes.	Profuse.
12.	Staphylococcus + staphylococcus serum[2] + leucocytes.	

In comparing these results, repeated study of the microscopic specimens showed that phagocytosis (1) is less in salt solution than in normal aqueous; (2) is more marked in immunized aqueous and then is about equal to that found in normal serum; (3) is still more marked in the 2d normal aqueous though most pronounced in the immunized blood serum.

[1] The aqueous humor of the immunized animal.

[2] The blood serum of the immunized animal.

CONJUNCTIVITIS AND PURULENT INFLAMMATION OF THE EXCRETORY DUCTS OF MEIBOMIAN GLANDS CAUSED BY AN ENCAPSULATED GRAM-NEGATIVE DIPLOBACILLUS.

By Dr. O. LANDMAN, Toledo, Ohio.

(With two figures on Text-Plate XXVI.)

On October 20, 1908, the patient presented herself for treatment. She complained of a feeling of sand in the left eye, and was greatly annoyed by the upper lid sticking firmly to the eyeball on awakening. She said that the lashes were glued together.

On inspection, muco-pus could be seen in the inner canthus, also in the lower cul-de-sac. The eyelashes were grouped into tufts by masses of yellowish inspissated pus. The margins of the lids were very red, but the conjunctiva showed only a slight hyperæmia. Muco-pus was found underneath the upper lid.

At this time, and for the following four weeks, at intervals, the conjunctiva was painted with a two per cent. (2%) nitrate of silver solution. The patient was given a solution of sulphate of zinc, one-eighth of one per cent., to be used three times a day.

About one month after the conjunctivitis had started, pustules formed in the mouths of the excretory ducts of a number of Meibomian glands.

The same symptoms continued without change, although the treatment was altered; and on March 12, 1909, five months after the trouble had begun, there was the same muco-purulent discharge, and the mouths of the Meibomian glands still showed pustules.

On trying to remove the muco-pus with a platinum loop, it was so very sticky and tenacious that it could only be caught up by twisting it around the loop.

Smears made from the muco-pus, and from the pus from the contents of the ducts of the Meibomian glands, showed

TO ILLUSTRATE DR. LANDMAN'S ARTICLE ON "CONJUNCTIVITIS AND PURULENT INFLAMMATION OF THE EXCRETORY DUCTS OF MEIBOMIAN GLANDS CAUSED BY AN ENCAPSULATED GRAM-NEGATIVE DIPLOBACILLUS"

FIG. 1.—Magnification, 1000 ×.

Conjunctival Secretion. Diplobacilli showing colorless capsules; simple stain From the original smear.

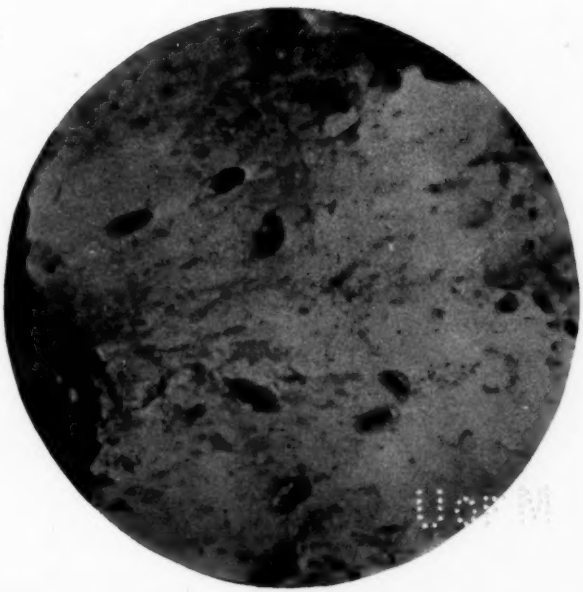

FIG. 2.—Magnification, 1500 ×.

Diplobacilli stained for capsules. Johne's Method. From the original smear.

a Gram-negative diplobacillus surrounded by a capsule. There were also some short chains present, made up of two or even four sets of diplobacilli. There were some shorter forms, probably younger, but almost always of the double type. They were all of about the same diameter.

On April 11, 1909, almost six months after the disease had begun, the same Gram-negative diplobacillus was demonstrable in the discharge, indicating that the treatment had been without avail. It was again isolated in pure culture, giving the strain No. 2 referred to later. The eye now showed the symptoms of a chronic conjunctivitis with the conjunctiva thickened in places, so that the markings of the Meibomian glands were hidden.

It had been noticed that, in acid bouillon the growth of the diplobacillus was very much interfered with and, accordingly, it was inferred that a weak acetic acid solution might cure the disease.

Following this, acetozone was recommended, because in solution, and upon standing, acetic acid is set free, and acetozone in itself is a disinfectant.

Therefore, the patient received for use a $\frac{1}{1000}$ solution of acetozone, which finally controlled the condition.

Thus, from a laboratory experiment, the remedy for the disease was found.

Two strains of *pure cultures*, agreeing in every respect, were obtained from the diseased eye; one, in the first beginning of the conjunctivitis; and the second, six months later.

Morphology.—The measurements were made of stained diplobacilli, from the original smears and from different culture media, no older than 48 hours.

The average length of a single bacillus, in the original smear, was 3.04μ, and width .96μ. In gelatine, length varied from .85μ to 3.25μ, and width from .64 to .96μ. In agar, engths varied from 1.65μ to 3.25μ, and width from .64 to .96 . They are fairly thick, large bacilli with rounded ends, bearing a pronounced capsule in the original material (Fig. 1). Exceptionally, as will be shown, capsules may form on certain culture media at a low temperature. There are some shorter forms and, at times, threads of four to six cells are met with. The diplobacilli are a trifle larger in the cultures than in the original material.

The capsule is as wide as the body itself (Fig. 2). The capsules are present in the original smears, and to a less

extent in the cultures from the mouse, rat, and guinea-pig.

Clear oval areas can be seen in unstained cultures from agar and in the diplobacilli recovered from the blood of inoculated animals.

Involution forms, showing slightly enlarged ends, are found in all of the cultures after 48 hours.

Staining.—The diplobacilli stain readily with the ordinary anilin dyes, but do not take the Gram stain—that is, they are Gram-negative.

The capsules are readily stained in the original material, and in the peritoneal exudates of the mouse, rat, and guinea-pig, but in other cultures are difficult to stain. In order to obtain capsules for staining, streak cultures were made on glucose-agar slants, which were kept in the ice-box, at a temperature of 10° C. for ten days, when the capsules were beautifully developed and readily stained by Johne's and Ribbert's methods.

The capsules, when present, are easily stained by the methods of Johne, Hiss (copper sulphate), Welch, and Ribbert; also by treating them with a gentian violet, one-half saturated aqueous solution, and washing in a one-fourth per cent. solution of potassium carbonate.

Flagella.—Loeffler's method and Zettnow's antimony tannate silver method showed no positive evidence of flagella, although at times heavily stained lines were obtained, which did not show the wavy character of true flagella and, consequently, were not regarded as such.

Long needle-shaped crystals appear at 20° C., room temperature, in agar slants in from three to ten days, and occasionally in older glycerine stabs.

Biology.—The diplobacillus is non-motile, although it possesses marked molecular movement. In acid bouillon the molecular movement is not so great as in growths from other media. It does not liquefy gelatine, is a facultative anaërobe, and does not form spores. It grows rapidly at 20° C. in almost any media, and luxuriantly at 36° C. In old sealed cultures, on plain agar, or on glycerine agar, after three and one-half months, it is still viable. Moist heat at 60° C. kills the diplobacillus in a few minutes. It grows slowly at a temperature of 10° C. in an ice-box.

GROWTH IN CULTURE MEDIA

Agar Plates.—At 36° C. after 18 hours. To the naked eye the surface colonies present a shining appearance, often convex, round, white in color, fairly thick and mucous-like. The deeper colonies are oval and have a yellowish color. There is no odor produced. Measurements of the surface colonies, at the end of three days, showed an average diameter of *2mm*, with a slight increase after eight days at 20° C. Under a low power the surface colonies are somewhat granular, with smooth edges; the deeper colonies are finely granular with smooth edges.

In Glucose-Agar stabs, at 20° C., the organism develops along the line of inoculation, while on the surface the growth spreads to the extent of 5 to *7mm*.

Glycerine-Agar stabs, at 20° C., show a similar growth along the length of the stab and on the surface; the latter forming a moist and glistening thick slimy cap. The growth is much more luxuriant than in glucose agar.

Serum-Agar slant, at 36° C. and at end of twenty-one hours shows a luxuriant growth, yellowish white, thick, and slightly spreading, with isolated colonies.

Plain-Agar slant, at 36° C., the growth is rapid and is recognizable after four hours; later becoming thick and yellowish white.

Gelatine Agar, at 36° C., the growth is slow, beginning to appear in about two days.

Dextrose Agar, at 36° C. after forty-eight hours, a very luxuriant growth, white and shining.

Glycerine Agar, growth is very thick, whitish, shining, mucous-like, after forty-eight hours, at 20° C., room temperature. The condense water of the agar slants is cloudy with a slimy precipitate.

Blood Agar (one part nutrient agar and one part defibrinated rabbit's blood, mixed and inclined), at 36° C., after twenty-four hours, a rich and abundant growth is obtained without change in the color of the hæmoglobin. The growth is moist and spreading with no liquefaction.

Glycerine Potato, at 36° (Roux tubes), after twenty-four hours, a luxuriant growth, yellowish white, moist, glistening honey-like, very slimy and tenacious.

Plain Potato, at 36° C., after twenty-four hours, a luxuriant growth, moist and glistening, of a light yellowish color.

Gelatine Plates, at 20° C., show a slow growth, after two days. They show no evidence of liquefaction, and no cloudiness of the gelatine.

Gelatine stab, at 20° C., a growth slowly develops along the stab, either continuous or more or less broken up into minute colonies, while on the surface it is slightly spreading and moderately convex. No change of color in gelatine, but occasional production of crystals. Streak cultures on gelatine show a decided growth on second day, yellowish white and glistening.

Glucose Gelatine stab, growth is more abundant than in plain gelatine; is more rapid, *with no gas production.*

Plain Litmus Gelatine, growth is slow and slightly acid.

Plain Milk is not coagulated at 36° C.

Litmus Milk, slight acid production in course of twenty-four hours, at 36° C.

Dunham's Solution, faint growth after twenty-four hours. *No indol is formed.*

Plain Bouillon, at 36° C., in four to six hours, a very fine cloudiness develops, which rapidly increases, giving rise, after twenty-four hours, to an abundant sediment which, after agitation, settles slowly.

In Acid Bouillon (+2.5 to +3.0), at 36° C., there is a slight growth in twenty-four hours, and the production of the slimy product is diminished.

Glucose Bouillon, in a closed fermentation tube at 36° C., gave abundant growth in the closed arm. *No gas production* after two days.

Pathogenesis.—To test the effect of the culture in animals, a suspension of the entire growth on a plain agar tube, in 2*cc* of normal salt solution was made. 0.5*cc* was given intraperitoneally to a white mouse, and 1.5 to a rat. Both died within twenty-four hours. Autopsy showed an exudate in the pleural cavity, congestion of the lungs, peritonitis with purulent exudate. Rigor mortis still present.

A second mouse was given .25*cc* intraperitoneally, of a similar suspension, and died within twenty-two hours. Autopsy findings were the same.

A young guinea-pig received, intraperitoneally, 1*cc* of a suspension of the growth on a plain agar tube, and died within twenty-two hours. It also showed an exudate in the pleural cavity, congested lungs, and purulent fluid in the peritoneal cavity.

A young rabbit received 1*cc* of the same suspension that was given to the guinea-pig, but it did not produce death.

The germ was recovered in *pure cultures* from all of the animals. It was always the same Gram-negative diplobacillus.

The injection of 0.5*cc* of a similar suspension killed a rat within twenty-four hours.

That a relatively large dose of the organism is necessary in order to kill a rat is seen from the fact that a mixture of 0.25*cc* heart's blood and a suspension of half of the liver of the first rat in sodium citrate solution (½ of 1%), when injected, intraperitoneally, failed to produce death. Two experiments of this kind were made with the same results. In mice direct passage was effected.

As indicated, rats showed less susceptibility than mice, since they required larger doses, and because of the impossibility of producing infection from rats previously inoculated.

Rabbits are apparently immune.

INOCULATION EXPERIMENTS OF RABBITS' EYES.

The eyes of three rabbits were inoculated by introducing suspensions of the cultures into the anterior chamber.

Two were treated thus with a bouillon culture; one of them proved negative, while in the other there developed a plastic iritis, which was attributed to trauma.

A third eye was inoculated with 0.25*cc* of a suspension of the growth on a plain agar tube, and the result was negative.

An intraperitoneal injection of this same suspension was given to a rat, which died in about twenty-four hours.

Agglutination Tests.—A rat received 0.5*cc* of an agar suspension, made as in former experiments, previously heated to 60° C. for ten minutes. Four injections were given within a period of thirteen days. The rat was bled in the afternoon

of the thirteenth day. The heart's blood was drawn into a tube, sealed, and placed in the ice-box. The blood was kept over night, and the serum, which separated after eighteen hours, was tested with the two strains of this organism and with Friedlander's pneumo-bacillus.

The following table gives the results of the agglutination tests with the first and second strains of the diplobacilli and Friedlander's bacillus with dilutions of the serum. The identity of the two strains, isolated from the patient, is thus shown; the second having been isolated six months after the first.

Dilutions.	First Strain. Minutes.		Second Strain. Minutes.		Friedlander's. Minutes.	
1–1	+++	10	+++	10	o	o
1–10	+++	15	+++	20	o	o
1–30	++	16	+++	45	o	o
1–50	++	21	++	55		
1–100	o	o	+	75		
1–200	o	o	o	o		
1–500	o	o	o	o		

+++ =Marked Agglutination.
++ =Moderate "
+ =Slight "
o =None.

The results of this investigation prove that this Gram-negative encapsulated diplobacillus is a new organism, and must be added to the list of pathogenic bacteria already discovered.

It is capable of producing a chronic and obstinate conjunctivitis, and was first discovered in the conjunctival sac, as described in this paper.

There are two other Gram-negative bacilli that produce conjunctivitis, from which this new diplobacillus must be differentiated, namely: Morax-Axenfeld diplobacillus and Friedlander's pneumo-bacillus.

According to some observers, the Morax-Axenfeld diplobacillus has a faint capsule, whereas this diplobacillus has a distinct capsule. Axenfeld says:

"The diplobacilli of the Morax-Axenfeld type grow, with positiveness, only at 36° C., and only on blood serum, serum-agar, or on media which contain ascitic or pleuritic fluid."

This diplobacillus grows at a temperature as low as 10° and on *all* media. The Morax-Axenfeld diplobacillus is non-pathogenic, whilst this one *is* pathogenic.

The characteristics which distinguish this diplobacillus from Friedlander's pneumo-bacillus are: that Friedlander's pneumo-bacillus is smaller and does not occur in pairs as constantly as this diplobacillus; Friedlander's diplobacillus produces gas and indol, this does not; the crucial test is, that suspensions of cultures of the Friedlander's pneumo-bacillus *do not* agglutinate with dilutions of the serum obtained from the blood of rats that were inoculated with cultures of the Gram-negative diplobacillus which is the subject of this paper.

It is possible that forms which have hitherto been called Friedlander's or Morax-Axenfeld diplobacilli, from a hasty clinical examination by staining a smear, may have been mistaken for this diplobacillus; and perhaps, in the future, culture methods may disclose this diplobacillus to be more frequently a cause of conjunctivitis.

This opportunity is taken to acknowledge the great aid rendered by Prof. F. G. Novy, of the University of Michigan, in the pursuance of this work; and also to his assistant, Dr. Frank McJunkin, for the help given during the progress of the investigation.

CHORIO-RETINITIS TUMIDA.

By Dr. CHARLES J. KIPP, Newark, N. J.

UNDER the head of chorio-retinitis tumida, Hirschberg (*Centralblatt f. praktische Augenheilkunde*, 1888, p. 162) has described two cases in which he found foci of chorio-retinitis raised to 1*mm*. To me these cases appear to have been cases of the form of chorio-retinitis circumscripta, so excellently described by Hill Griffith (Norris and Oliver, *A System of Diseases of the Eye*, vol. iii., p. 355), in which a deposit on Descemet's membrane and opacities in the vitreous nearly always accompany a patch of varying size of bluish white or yellowish white color in some part of the retina, and which leaves a conspicuous area of atrophy of the choroid, fringed or sprinkled with pigment. In Hirschberg's cases syphilis was supposed to be the cause of the eye disease. Such cases are seen frequently and in many a distinct elevation of the white patch can be made out in an early stage of the disease.

Schöbl in his article on "Diseases of the Retina," in Norris and Oliver, *A System of Diseases of the Eye*, vol. iii., page 478, refers to somewhat elevated plaques of chorio-retinitis with secondary syphilis. He says: "Once I found a large yellowish red focus near the optic nerve head. It was from four to five disk-diameters in size and raised a little more than one millimetre. It had an incomplete pigmented margin. In one eye of a case which I afterwards dissected, I found the papilla paler than normal and of a dirty yellowish white tint. In the temporal region between the papilla and macula and covering the latter there was a flat detachment of the retina, three disk-diameters in size. Here the retina appeared bluish gray in tint and was opaque. In the equatorial region I found several smaller yellowish red plaques of a prominence

of about one half millimetre each. The pigment epithelium in this region and near these plaques was altered. In some places it seemed wanting, while in others it seemed massed into black spots similar to those seen in simple disseminate choroiditis. Except on the parts close to the papilla, the retina was not in the least dim." The blood-vessels appeared normal. There were no hemorrhages. In all of his cases there were opacities in the vitreous body which consisted mostly of fine dust-like bodies. In some cases larger flakes could be seen in the posterior parts of the vitreous body. With regard to the diagnosis he says: "If the objective and subjective symptoms are well considered, the diagnosis is generally made without any difficulty, even without anamnesis and other concomitant symptoms of secondary syphilis. It might perhaps be confounded with a beginning choroidal sarcoma or with coalescing tubercles of the choroid. The objective condition of the nerve head and the subjective retinitis symptoms should however prevent any error in diagnosis even when the personal history is wanting or purposely falsified. The prognosis, he says, must be termed a favorable one, since the ones that he has seen as well as those of Hirschberg all terminated in a relative cure. He advises anti-syphilitic treatment.

In the case below reported the elevation of the chorio-retinitis foci was well marked, while the media were perfectly transparent. There was no deposit of precipitates on Descemet's membrane and the vitreous was perfectly clear. Moreover the swollen part of the retina was in the following case opaque only in spots and lines.

The patient Mrs. A. R., thirty-four years of age, is a short stout woman, in apparently robust health; she is the mother of two children who are in good health, and has had one miscarriage. She has never been seriously ill, has never had rheumatism, and denies having syphilis. Her father and mother are dead. The father was eighty years old at the time of death. The mother died of apoplexy at the age of sixty-four. The patient has two sisters and two brothers who are all free from eye disease and in good health. Up to five days before she came to the Newark Eye and Ear Infirmary her eyes had never given her pain and vision had always been good in both eyes.

She was first seen on December 9, 1907. She complained

of pain over the right eye and stated that five days previously after a fright the vision of the right eye suddenly became blurred and at the same time she had pain of a neuralgic character in the right half of the face and in the right ear, together with a numbness of the skin of the right cheek, and these symptoms have continued since. She was otherwise in excellent health. A careful examination of the right eye on this day made by Dr. Webner showed the *right* to be emmetropic. V = ¾. No apparent disease of the right eye. The eye ground was perfectly normal. The eye was put under a solution of homatropin 3%. Shadow test showed H. 0.75 D., and she accepted this glass. The case was diagnosed as supraorbital neuralgia and she was given quinin. Shortly after this examination she moved away from Newark to a place nearer New York, and as her eye continued to trouble her, she consulted an ophthalmologist of large experience in New York City, who, as she says, after repeated and long-continued examinations of her eye, told her that she had a tumor in her eye and advised immediate enucleation, and as she was unwilling to submit to the removal of her eye, he told her that he would not have anything further to do with her.

She returned to us on January 15, 1908, when, to the amazement of the surgeons who had examined her five weeks before, the following picture was presented by this eye. The media were perfectly clear; the disk was of normal color and its outline was distinct on nasal side and above and below; on the temporal side right at its very edge, the retina was raised abruptly to 1*mm* (3 dioptries) and was slightly milky. The raised area extended above somewhat higher than the upper margin of the papilla, and also somewhat below the lower margin; towards the macula it extended somewhat more than the breadth of the disk. It rose abruptly on the margin of the disk, and gradually sloped to the level of the normal retina outward, upward, and downward. The swelling was of the shape of an olive. The opacity of the retina hid the vessels in places. There were numerous white silvery spots and lines in it and a few extravasations of blood in different parts of the swelling. The rest of the retina was perfectly transparent. Tn. I was asked to see the patient at this time and found the condition above described. The swelling certainly looked a good deal like a new growth, but the fact that a retinitis was present made me doubt that the swelling was caused by a growth beneath it. I have had the opportunity of seeing sarcomata of the choroid in their earliest stages and in not one of these did the retina show signs of inflammation. It seemed to me more probable that an exudate from the choroid was push-

ing the retina forward, and I therefore advised against enucleation and suggested that she be given potassium iodide. From this time on, I saw the patient almost every week and examined her most carefully each time. For some weeks the symptoms of retinitis increased, new silvery white lines and shining white dots appeared, especially in the direction of the macula, and new extravasations of blood were noticed in the periphery of the swelling. The swelling itself seemed to be somewhat larger and its height greater than when first seen. For some months this condition remained about the same. About six months after we first saw the swelling, it began to decrease in height and the silvery spots and lines became absorbed. Now, fifteen months after she first came to us and thirteen and a half months after we first saw the swelling, the retina in the region formerly raised is on a level with the disk and the rest of the retina. The retina is nearly transparent here, and beneath it is a very large patch of very dark pigment. Between the disk and the macula the pigment epithelium is irregularly distributed. The disk is somewhat whiter in the temporal half than elsewhere. Vision is reduced to $\frac{4}{15}$. The visual field is of normal size, but the blind spot is much enlarged inward and upward. Feb. 12, 1909, V = $\frac{4}{15}$.

The outcome leaves no doubt that this was a case of circumscribed chorio-retinitis situated close to the disk, or, to use the name, recently suggested by Jensen of Copenhagen for such cases, Retino-choroiditis juxtapapillaris (Graefe's *Arch. f. Ophthalmologie*, lxix., Heft 1, p. 41).

I have seen a number of cases similar to the one here described. In one case briefly mentioned in the discussion of Dr. Hubbell's paper (*Transactions* of the Ophthalmological Section of the American Medical Association in 1906, p. 86), the focus was situated very close to the margin of the papilla, and the papilla itself participated in the inflammation, giving it the appearance of a choked disk with a swelling amounting to three dioptries. After the disease had run its course, an atrophic patch of choroid adjoining the margin of the disk was the conspicuous evidence of the nature of the morbid process.

In a case very recently seen by me and still under observation in a woman forty years of age, the disk was greatly enlarged and swollen, and the changes in the retina extended for some distance in all directions, and now that absorption has

taken place the focus is seen to have been situated near the upper margin of the disk.

In a case similar to the one above fully reported in which the focus was situated on the nasal side of the disk and the swelling of the retina was not as great, the patient, a woman thirty-five years of age, admitted that she had had a primary syphilitic sore five years before, for which she was treated by a competent physician. She had no other symptoms of syphilis at the time the chorio-retinitis developed, and it is very doubtful in my mind that the syphilis was responsible for the eye disease. She recovered with an absolute scotoma corresponding to the seat of the lesion.

In all my other cases, I was unable to find in the patients' general health a cause for the eye disease. A careful examination made by competent physicians failed to show disease of any organ or of the accessory sinuses of the nose; blood examinations were made in several such cases, but they were negative.

I look upon these cases as being primarily a circumscribed choroiditis in which the retina over the focus became raised and involved in the morbid process by an exudation from the choroid. It is possible as suggested by Jensen (*loc. cit.*) that the starting point of the disease may be found in the anterior part of the optic nerve sheath.

In my cases partial recovery of vision occurred under the use of iodide of potassium, but whether this remedy was really of much assistance must remain an open question.

REPORT OF A CASE OF TRANSIENT CYCLOPLEGIA DUE TO GLYCOSURIA.[1]

By Dr. W. H. ROBERTS, Pasadena, Cal.

PARALYSIS of accommodation, due to diabetes, is sufficiently rare to warrant the report of the following case:

Mr. W.——, aged fifty-one, first consulted me, March 30, 1908, concerning a tickling in his throat, which had troubled him for about two weeks. He was coughing a great deal, he felt and looked sick, and he had recently lost flesh.

Examination showed that his uvula was thickened and elongated, fauces congested; right cord somewhat immovable, irregular in outline, and reddened near the arytenoid cartilage.

Fearing that a tuberculous process might be commencing in his lungs, I advised him to consult a general physician for a physical examination. He placed himself under the care of Dr. Joseph D. Condit. Dr. Condit reported to me that there was no evidence of tuberculosis, but that his urine contained 7% of sugar.

Under appropriate diet, the sugar began to lessen, so that by the 7th of April, it was down to 5⅛%, and his weight, which was 133 pounds, began to increase. By the 29th of April, all sugar had disappeared from his urine, and, when last observed by Dr. Condit on the 22d of July, his weight had increased to 142 pounds.

On the 15th of April, when he had been under Dr. Condit's care for two weeks, his urine showed only ⅛% of sugar. At that time, he came to me again, saying that for a few days past he had been unable to read with his glasses, which had theretofore been perfectly satisfactory, and that he now needed them to see with in the distance. Until he noticed

[1] Read before the California State Medical Society, San José, April 20–22, 1909.

this failure of his glasses for reading his vision for distance had been perfect; but now he could not see in the distance without the use of his old reading glasses.

I found that he had been using for reading a pair of + sph. 1.75.

At that time

O. D. V. $= \frac{6}{22.5}$

O. S. V. $= \frac{6}{15}$

Manifest examination showed:

O. D. + sph. 1.75 $= \frac{6}{6}$

O. S. + sph. 1.75 $= \frac{6}{6}$ $1\frac{1}{2}°$ esophoria.

For reading at thirteen inches, he required to be added to the above + sph. 2.25.

External examination of the eyes showed everything normal; pupils were of normal size, and they reacted to light and accommodation. Ophthalmoscopic examination showed the media clear and the fundi normal. A test on the following day confirmed this examination, so I ordered the foregoing lenses for him.

On the 18th of May, he came in again very much improved in his general health. He said that until within a few days, these last glasses had been perfectly satisfactory; but that now he could not see so clearly in the distance with them, and he found that he had to hold newspapers and books too close to his eyes to read with comfort. His vision with his distance glasses now was only $\frac{6}{12}$.

Manifest examination showed:

O. D. V. $\frac{6}{15}$ + sph. 1. $= \frac{6}{6}$

O. S. V. $\frac{6}{9}$ + sph. 0.75 $= \frac{6}{6}$

with + sph. 2.25 added for near, and these I ordered.

Shortly after this Mr. W.—— returned to his home in the East. In a letter, dated Feb. 8th of this year, he wrote me that his eyesight was very much better than when he was in Pasadena last year. His accommodation continued to return so that it was necessary for him to go back to the original reading glasses which he was using when he first came to me. A few days before writing to me he had broken his old lenses, and, for a short time, he had made use of the ones I had last given him, which brought his near point within eight or ten inches of his eyes. His general health has been excellent, though he is still very careful in his diet.

Dr. Geo. M. Gould, in an article on "The Refraction Changes Dependent upon Glycosuria" (1), has collected twenty-two cases in which changes have occurred. These he has ar-

ranged in three groups: the first consists of six cases, and shows an increase of myopia; the second is made up of eight cases in which hyperopia is diminished, which is a change similar in character to those in the first; the third is composed of eight cases in which an increase of hyperopia is noted.

We are not concerned with the first and second groups; but in the third group we are interested, for the case just reported seems to belong to this class. In all eight cases, an increase of hyperopia is noted; and, in seven of these eight cases, it disappears, returning to the original amount with the subsidence of the sugar.

As an increase of myopia is the most common refraction change occurring in diabetes, and as fourteen of the twenty-two cases reported in Dr. Gould's article had such changes, Dr. Gould is inclined to belittle the findings of the men who report the eight cases of increased hyperopia, claiming that the observations are unreliable, inasmuch as a mydriatic was used in but one case, that of a boy of seventeen. The ages of five of the remaining patients are forty, fifty-five, sixty-eight, fifty-one, and fifty-three, in each of whom there could have been but slight latent hyperopia. Undoubtedly, in each of these eight cases, there was a paralysis of accommodation; although such an explanation for the increased hyperopia does not seem to have occurred to Dr. Gould, who says: "When eight reporters find such an illogical, if not impossible, reversal of the natural consequences as testified to by fourteen, it behooves us to doubt the accuracy of the oculists' tests and reports, rather than to indulge a belief in the inherently improbable and impossible."

Delord and Revel (2) report a case, very similar to mine, in a woman forty-eight years of age. She had a sudden bilateral paralysis of accommodation, without involvement of the sphincter pupillæ. With the disappearance of the sugar from her urine, she regained her accommodation. With Schmidt-Rimpler, these authors agree that cycloplegia from diabetes is rare, which is contrary to the opinion held by Wecker and Landolt.

Of particular interest to me in the history of my patient, was the fact that his cycloplegia did not appear until after his health had begun to improve, when the sugar had de-

creased from 7% to about ½%, showing that possibly a similar toxin to that causing a post-diphtheritic paralysis caused the lesion.

The prognosis in diabetic cycloplegia seems very favorable, as, in the majority of cases, under proper dieting, the sugar can be eliminated from the urine, and the ciliary muscle regains its function.

REFERENCES.

1. GOULD. "The Refraction-Changes Dependent upon Glycosuria." *Medical Record*, April 20, 1907.

2. DELORD and REVEL. "Paralysis of the Accommodation in Diabetes." *Archives d'ophtalmologie*, xxvii., p. 764.

To Illustrate Dr. Jameson's Article on "Re-Attachment in Iridodialysis: A Method which Does Not Incarcerate the Iris

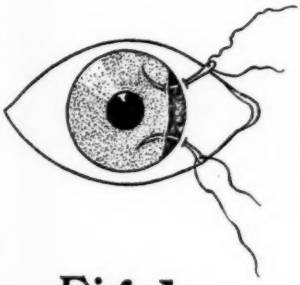

Fig. I.

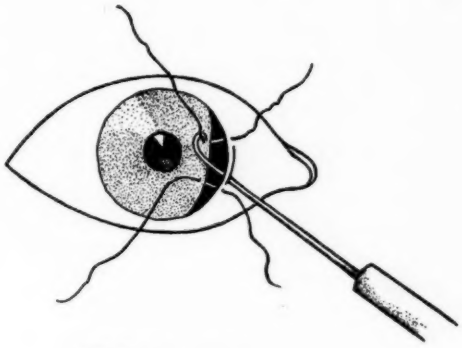

Fig. II.

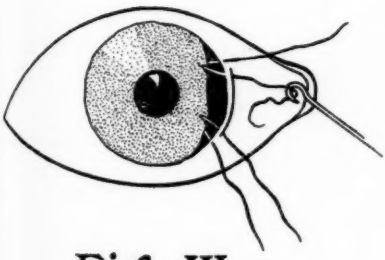

Fig. III.

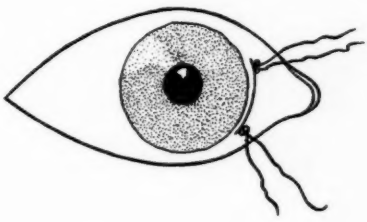

Fig. IV.

RE-ATTACHMENT IN IRIDODIALYSIS: A METHOD WHICH DOES NOT INCARCERATE THE IRIS.

By Dr. P. CHALMERS JAMESON, Brooklyn, N. Y.

(With 4 illustrations on Text-Plate XXVII.)

IT is with some hesitation that I present this operation for the cure of iridodialysis, knowing that such operations have not been regarded with favor, owing to the danger of cyclitis, and possible sympathetic irritation. Perhaps this contingency has been enhanced by the fact that operations for the re-attachment of the iris have for the most part depended upon an incarceration of a portion of the iris in a wound made for its reception, in the corneal margin. This operation does not incarcerate the iris, but brings the torn surface in contact with a freshened surface, within the anterior chamber. That the operation as described hereafter will be followed by no untoward results can only be determined by a larger number of cases than the four which the writer can at present report.

Its aims are: 1. Re-attachment of torn iris as near to its normal anatomical position as possible. 2. Re-attachment to a fresh surface within the anterior chamber without incarceration. 3. Simple, accurate, and practical technique. The first—re-attachment as near anatomically correct—is accomplished by making the necessary incision into the sinus of the anterior chamber well back from the limbus. The second,—re-attachment without incarceration,—by bringing the torn iris against a linear incision within the anterior chamber, *not between its lips;* and the third,—simple and accurate technique,—by the introduction of sutures before incision is made into the anterior chamber, and by impinging

the iris against the resistant posterior surface of the cornea the iris is neither torn in the effort to perforate it nor lacerated by forceps. The following description of the operation will be understood by glancing at the diagrams.

The sutures are introduced, before any incision is made into the anterior chamber, in the following manner. The needle penetrates the sclero-corneal margin, two or three *mm* back from the limbus (Fig. 1). It is carried into the anterior chamber, under the torn margin of iris, engaging the least possible portion, less than 1 *mm*. This is to avoid strangulation of the circulus major as well as the preservation of tissue. The torn edge of the iris is impinged against the posterior surface of the cornea by the point of the needle, and both cornea and iris are perforated simultaneously, the needle being drawn through the cornea on the outside and liberated from the suture (Fig 2). The bend of horse-hair now contains within its curve, from point of entrance to exit, conjunctiva, sclera, and the edge of torn iris and cornea. Two things remain to be accomplished. First, to exclude this bridge of cornea from the structures to be approximated and tied, and second, to provide a surface freshened within the anterior chamber, to which the torn circumference will agglutinate when contact is brought about. The incision as illustrated in Figs. 2, 3, 4, serves in this dual capacity. It should be made in a straight line between the points of introduction of the sutures. The ends of the incision should have a short bridge of sclera and conjunctiva between them and the point of entrance of the needle or needles. A hook is now introduced into the anterior chamber (an iris hook will do) and guided in front of the iris between it and the posterior surface of the cornea (Fig. 2). It engages in its bend that portion of the thread extending from the anterior surface of the iris to the posterior surface of the cornea. This is pulled upon with great care, slight pressure being made on the posterior surface of the cornea rather than on the iris, and the end of the suture which passed through the cornea is now brought through the anterior chamber and thence between the lips of the incision (Fig. 3), thus liberating the corneal bridge and enabling the suture to be tied without including the cornea. The sutures, when tied, bring the torn circumference of the

iris in contact with the inside of the linear incision and to the points of the primary perforation of the needles, but not between the lips of the incision. The re-attachment of the iris after agglutination has taken place is thus accomplished. In moderate iridodialysis, one suture is all that is necessary, but if two be required, they should both be introduced into the iris before either is tied, as efforts at introduction after the iris has been attached in part is apt to result in tearing it away again. The needle should be very keen, slender, and with a larger curve than would be indicated by the path of tissues to be traversed. The suture material most desirable is horse hair or very fine silkworm gut, for the reason that they are the materials which glide easily through the iris. Failure resulted in one case by reason of a silk suture ruffling and twisting the iris upon itself.

The following accidents may happen. If the suture material is not suitable (horse hair or silkworm gut), the suture may ruffle and in drawing it through, the iris may roll upon itself, rupture, and be destroyed. Smooth suture material should always be selected. If two sutures are introduced and one is tied before the introduction of its fellow, the first may be torn out in the effort to introduce the latter. If the ends of the incision be brought too close to the path of the sutures, or should happen to open into their channel of entrance, there will be no bridge of tissue for anchorage after they are tied (see Fig. 3). If the needle is not selected with care as to size or curvature, too large a portion of the iris may be included in the suture, or the iris may be ruptured in endeavoring to impinge it against the posterior surface of the cornea at a point farther away from the torn edge than the elasticity of the iris or its ability to stretch will allow. The sutures may be left three or four days. The perforations of the cornea by slender and sharp needles is unattended by any untoward result, and the impingement of iris by the point of a needle against the posterior surface of the cornea enables one with minute accuracy to obtain the desired portion of iris in the bend of the suture without tearing or lacerating. The advantage of bringing the torn iris in contact with the fresh surface of an incision within the anterior chamber or sinus is evident. There is no incarceration, therefore less liability

to sympathetic condition, and the normal relationship of the tissues is preserved to the greatest degree. There is greater preservation of iris tissue, therefore less likelihood of displaced pupil. The danger of nerve irritation or circulatory disturbance is reduced to the minimum, as the portion of iris held in apposition to the wound is less than 1*mm* and is released on the third day. Two or even three incisions may be substituted for the single, illustrated in Fig. 3, and if the detached periphery is large this is advisable, as the large incision might predispose to prolapse. The following is a short résumé, with observations on the four operations done.

CASE 1.—Wm. T., referred by Dr. Frank Little, at Brooklyn Eye and Ear Hospital, Nov. 22d, 1905. The operation was performed three months after iridodialysis was brought about. The iridodialysis was large, about 8*mm* in length. One fine silk suture was introduced by the method described, corresponding to a point a little lower than the centre of the torn iris. The operation was accomplished without incident and the suture tied, bringing the iris in contact with the inner surface of the wound.

This patient made an uneventful recovery, all reaction quieting down two and one half weeks from the time of operation. Firm agglutination of the iris to its new attachment was accomplished and a good normal pupil which reacted to light and accommodation obtained. The rupture being large and having no previous experience with operation, one suture alone was introduced, when two were necessary to re-attach the iris in the whole length of the tear. The result, after healing, was a small unattached area, which was the subject of operation three months later.

CASE 2.—In this case, silk again was used and an untoward happening occurred in consequence. In drawing the suture through the iris, the silk ruffled, twisted the iris upon itself, pulled away again the iris which had already been re-attached at the previous operation, and, as a final result after tying the suture, I succeeded in re-attaching the iris to the circumference, but not producing the best cosmetic result, as it was slightly torn and its anatomical relationship twisted.

This case proved valuable in inaugurating better technique.

1st. Twisted silk or braided suture was discarded for smooth horse hair or fine silkworm gut, which would glide uninterruptedly and smoothly through the iris and not ruffle. 2d. When more than one suture was required, it was thought advisable to have them introduced in the iris previous to making the incision into the anterior chamber and before attempting to tie either (Figs. 1 and 2). By so doing the danger of tearing out a suture already tied by the slight stretching of the iris incident to the introduction of another is avoided.

CASE 3.—Charles C., operated upon at the Brooklyn Eye and Ear Hospital, on Oct. 21st, 1908, illustrates that after seven months, under the most adverse conditions, an iris can be re-attached by this procedure. The iris in this case was not only detached from its circumference, but evident nutritive changes between the points of detachment had taken place. The iris between these points was atrophied and translucent, its rigidity was lost, and it hung down over the pupillary centre. The detached border had fallen over in front of the pupillary margin, reversing the normal anatomical position. The injury had been accompanied with traumatic cataract, which, after absorption, left what would have been a good pupil, if unobstructed by this blemish. This was not a selected case, but the operation accomplished the object of drawing out of the pupillary zone a flaccid, atrophied iris, which cosmetically and visually was out of place. It now hangs from the upper circumference border. The upper lid covers it, which was not the case before, and the boy has a black pupil.

This case demonstrates that an iris which has lost most of its anatomical constituent can, seven months after injury, be re-attached and retained in position, at the point of suture, and established the belief of the writer that normal iris tissue can be re-attached at any time, by this method.

CASE 4.—Operated upon at the Brooklyn Eye and Ear Hospital. Mr. A., Oct. 28th, 1908, three weeks after injury, iridodialysis moderate in size. Two horse-hair sutures introduced, one accidentally withdrawn before tying. The operation was successfully performed. Reaction was not marked and subsided in three weeks. After re-attachment had taken place, the pupil was slightly displaced, but not more than would be accounted for by loss of tissue at the circumference of the torn iris. A slight cleft at the circum-

ference remained, which would not have been the case if two sutures had been employed.

I mention the difficulties experienced in these operations, believing that they, as in all new operations, are of most interest, but with these guarded against and with the improved technique obtained and profited by experiencing them, the operation is a simple one and easy of accomplishment. The operation is naturally followed by inflammatory reaction, but not more in the cases operated upon than would be present in a mild and transient iritis.

The question has been asked, Will the torn edge of iris agglutinate itself if the iridodialysis is not of recent occurrence? The writer believes that it will any time after the original injury, as the plastic exudate thrown out from the iris at the point of contact is sufficient to accomplish it. All the four operations done were cases in which the injury had preceded the operation from three weeks to seven months. Again: Is one justified in advocating the operation from the cosmetic and visual standpoint? It is undoubtedly an improvement cosmetically, and to be relieved from the monocular diplopia and general disturbance in vision is desirable; but again, Is the risk of an operation considered among the subtleties of ophthalmic surgery too great? Dr. Eugene Smith, in his four cases operated upon after his modification of Critchett's operation, in which the smallest possible portion of iris is dragged into a small peripheral wound, made for its reception, reports no sympathetic condition. The operation, as before described, still further removes this possibility, as the iris is not dragged into the wound, but held in apposition and contact with it. The operative risk from the technical standpoint is nil, if delicately performed, and in the four cases operated upon there has not been a suggestion of sympathetic irritation.

VISUAL DISTURBANCE DUE TO THE USE OF HAIR DYE CONTAINING ANILIN.

By Dr. E. BERGER, Paris.

Translated from the German Edition, Vol. L., No. 4, by Dr. Matthias Lanckton Foster.

ONLY occasional observations of injuries to the vision from anilin poisoning in workers in anilin dyes, or anilin dyed fabrics, have hitherto been recorded. Uhthoff mentions only the cases reported by Marchesi and Veasey in which there was a concentric contraction of the visual fields and a reduction of the visual acuteness. In Veasey's case there was also a bilateral central scotoma and slight neuritic changes on the papilla. Galezowski speaks of the occurrence of photophobia and dimness of vision with ciliary injection among workers in anilin. MacKinlay reported a case of anilin poisoning in a workman forty-four years old, in which there was a brownish discoloration of the conjunctiva and cornea together with a slight loss of visual acuteness. Improvement began as soon as the patient discontinued his work. In Litten's case of poisoning with nitrobenzol accidentally mixed with anilin, the conjunctiva was stained violet and there were hemorrhages in the retrotarsal folds. There was also an intense violet stain of the fundus, while the papilla arteries and veins were deep black. The veins were dilated and there were occasional hemorrhages in their neighborhood. There was no loss of vision and no anomaly of the color sense.

The case I am about to report differs clinically from all those hitherto known and is besides of special interest from the standpoint of public hygiene.

Mrs. S., thirty-six years old, belonging to the well-to-do

class, consulted me Jan. 29, 1904, because she had noticed for several days that she did not see well with her left eye. She was of medium size, had always enjoyed good health, was the mother of two healthy children, twelve and ten years old. She had suffered from no general disease, influenza, rheumatism, or menstrual trouble, which could be ascribed as the cause of her visual disturbance. The central vision of her left eye was $\frac{2}{100}$, that of the right $\frac{10}{10}$. The visual fields were normal. There was a central positive scotoma of the left eye. The patient stated that she saw all objects with that eye as if through a veil shaped like a skull. The vertical diameter of the scotoma was from 10° above to 10° below the point of fixation, the horizontal from 10° nasally to 18° temporally, and therefore included the blind spot. Red and green were not recognized in the nasal part of the scotoma, but were recognized, though with difficulty and hesitation, in its temporal part, where the cloudiness was less intense. Objectively there was nothing abnormal about the lids, conjunctiva, and anterior portion of either globe. The pupils were moderately dilated and reacted normally to light, accommodation, and irritation of the skin. Ophthalmoscopically the left papilla was reddened, the right normal.

I placed the patient in a partially darkened room and prescribed salicylate of soda and linden-flower tea to promote free perspiration.

On the occasion of her next visit, Feb. 3d, the patient informed me that she had been using a hair dye on account of some gray hairs, and that headache and the visual disturbance of the left eye came on soon after the dye had been applied. I caused a chemical examination of this hair dye to be made, found that it contained anilin, and cautioned her against using it any more.

At this time the patient said that the scotoma was somewhat more transparent, though it had extended farther to the nasal side. The vision was only slightly improved, $\frac{1}{4}$. Her menses had appeared, and she had a menstrual œdema of the lids of each eye, particularly marked in the left lower lid. Her physician was much disquieted by this, and caused an examination to be made of her urine for albumin and sugar, which gave a negative result. Medication was discontinued and the patient ordered to bed for several days.

On *Feb. 8th*, the patient was much better. The œdema had disappeared from the lids, the vision of her left eye

was $\frac{1}{10}$, the scotoma was smaller and seemed to the patient to be broken through by bright points in several places. Colors were correctly recognized in all parts of the scotoma.

The rapid improvement of the trouble in the optic nerve may be explained by the theory that toxic substances, in this case anilin or its derivatives, are excreted by means of the menstrual hemorrhage. Charrin and Roche claim in fact that toxic products of metabolism are eliminated in this way.

The patient was directed to continue in a darkened room and to take one gram of potassic iodide daily.

On *Feb. 20th*, every trace of the scotoma had disappeared and the vision was 1.

Certain anilin colors alone produce poisonous effects, while others do not, as is shown by Litten's case, in which the general symptoms were produced by the nitrobenzol and, in spite of the great staining of the tissue of the eye by the anilin, there was no disturbance of vision. But a number of anilin colors have an intense toxic action. A. Gautier, in an exhaustive work on the chemical constituents of hair dyes, mentions among others the toxic anilin colors, particularly paraphenylendiamin (derivate of anilin). During the last six years numerous cases of poisoning by hair dyes have been reported in the Paris Society for Dermatology and Syphilis, and it is to be understood from Gautier's review of these cases that in many a single application of a cosmetic containing paraphenylendiamin was sufficient to induce a toxic action. This was illustrated by Brocq's case in which an attempt was made to dye a lock of gray hair. Although this lock of hair was afterward cut off, a general cutaneous affection persisted for two and a half months as a result. In several cases the poisoning resulted in death. In many cases the dye did not come into direct contact with the skin, as in those where poisoning was observed after the application of anilin preparations to yellow shoes for the purpose of staining them black.

The symptoms of intoxication with paraphenylendiamin and other toxic anilin colors may be divided into three groups.

1. Toxic skin eruptions, eczema, urticaria, with great burning and itching.

2. Gastro-intestinal symptoms, such as nausea.

3. Nervous symptoms, sleeplessness, dizziness, weakness of the legs, epileptiform attacks, and syncope.

When the cases are excluded in which visual disturbances were occasioned by a brownish-yellow discoloration of the cornea, the symptoms of amblyopia in anilin poisoning belong to the third group, that of diseases of the nervous system, and this appears to be, as claimed by Uhthoff, a toxic peripheral neuritis of the optic nerve.

In the case which came under my observation this disease of the optic nerve was only slightly marked, the vision of only one eye was affected, and the disturbance consisted of the appearance of a positive scotoma with a zone in which green and red could not be recognized. In several cases, like Veasey's, there was also a contraction of the visual field. The presence of a peripheral toxic neuritis was indicated ophthalmoscopically in my case only by the appearance of a hyperæmia of the optic nerve, while in Veasey's there were slight neuritic signs on the papilla.

The behavior of the pupils varies in different cases. Leloir observed mydriasis, Mueller and Smith myosis. In my case, as in the majority, there were no pupillary anomalies. These variations have not yet been accounted for.

This case is the first to be reported in which visual disturbance has resulted from the application of a hair dye containing anilin. It is probably not a unique case, it is more likely that the true cause has many times been overlooked. Dr. Eudlitz, who has made extensive investigations regarding diseases of the hair under the guidance of Prof. Fournier, informs me that the use of hair dyes is common in both sexes, and that diseases which should be ascribed to the toxic substances contained in them are frequently diagnosticated erroneously, as in the case of a woman thirty-two years old who was suffering from a toxic dermatitis due to this cause, but had been treated by her physician for facial erysipelas.

As a matter of public hygiene it would be well to prohibit the use of toxic anilin colors in the manufacture of cosmetics.

REPORT OF THE PROCEEDINGS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

By Mr. C. DEVEREUX MARSHALL.

An ordinary meeting of the Society was held at the Medical Society's rooms, London, on Thursday, May 6, 1909, Mr. R. Marcus Gunn, F.R.C.S., President, in the Chair.

Major R. H. Elliott, I.M.S., exhibited instruments: (*a*) an **evisceration spoon;** (*b*) an **optic nerve hook** for use in the operation of optico-ciliary neurectomy; (*c*) an **elevator** for use in extirpation of the lachrymal sac. Mr. Sydney Stephenson showed a case of **congenital anterior staphyloma** in a baby, who also had absence of patellæ, genu recurvatum, cryptorchidism, and other abnormalities. Mr. G. Winfield Roll showed a case **with unusual appearance of the optic disk** the result of a previous papillitis. Mr. E. Treacher Collins showed a case of **nodular leprosy affecting the eyes;** also a case of **progressive bilateral ptosis.** Mr. Holmes Spicer expressed the opinion that the condition was congenital, and thought the ptosis was closely allied to that in the cases of myasthenia gravis which had been described. Dr. G. Carpenter read a paper entitled **A clinical contribution to the study of tubercle of the choroid.** The first patient, under Dr. Porter Parkinson, was seven years of age, and had been getting thinner. Was admitted to hospital with physical signs at the right apex, front and back. There was a history of abdominal pain, but no cause for it could be discovered by palpation. The child then became drowsy and comatose, with great wasting. There were six choroidal tubercles, and flecks of retinal pigment were scattered about them. Three of the tubercles

were crossed by retinal vessels. A girl two years of age had been ailing for six weeks, and had had sickness and headache five days. When admitted, she was suffering from meningitis, and died on the sixth day. The right fundus was normal, and no tubercles or optic neuritis could be seen, but on the left side there was slight papillitis. Close to the optic papilla was a tubercle of considerable size, and several blood-vessels crossed its face. The retinal arteries were small, nothing was noticed about the veins. The surface vessels of the brain were congested. The diagnosis was verified at the autopsy. The next case was a boy æt. seventeen months, who had much the same physical signs. He had meningitis which was later corroborated by lumbar puncture. Another case was that of a boy æt. two years, who had had a bad cough five weeks, and when admitted had consolidation at both apices. There was a small amount of papillitis in both eyes. He died twelve days after admission, no changes having occurred in the fundus in the meantime. Several other cases of the kind were recorded, and the author then proceeded to discuss the readiness which had been shown to attribute such appearance to syphilis, whatever evidence to the contrary might have existed. He urged early and systematic ophthalmological examinations, which would often render unnecessary spinal punctures, or injections of substances to induce reactions. The paper was discussed by the President and by Dr. FREDERICK E. BATTEN, the latter gentleman commenting on the comparative infrequency of tubercle of the choroid in tuberculous children, and stating that he was under the impression that spinal puncture afforded a more certain indication than ophthalmoscopic observation. Dr. CARPENTER replied.

Messrs. MCKENZIE DAVIDSON and ARNOLD LAWSON read a paper entitled a case of **Spring catarrh treated and cured by radium.** The patient was a boy æt. twelve, who suffered from chronic photophobia, lachrymation, and slight conjunctival discharge, which had persisted for nearly a year, but no exciting cause could be discovered. Both tarsi were covered with dense hard excrescences, closely aggregated, and separated by deep narrow fissures. The retrotarsal tissue was swollen and hypertrophied, and the viscid discharge was spread over the conjunctival surface. The disease was con-

firmed by examination of the discharge. The irritability was chiefly marked in the morning. He was ordered complete cessation from close work, and dark glasses for out of doors. Yellow mercuric oxide was used, but no benefit resulted, and it caused too much pain. A week later he had cupric sulphate drops, of a strength of one grain to the ounce. In August the boy went to the seaside against his, Mr. Lawson's, advice, and got worse there. Bicarbonate of soda and hydrocyanic acid gave relief, and weak copper sulphate drops were used from time to time still. He then consulted with Mr. McKenzie Davidson as to the possibility of treating the case with radium. It was carried out for a year, during which period each eye was treated eight times. No pain or other immediate effect was produced, but the granulations gradually subsided. After the eighth application he was quite cured, but the interval before reading the communication was to make quite sure there had been no recurrence for a long time. Thirty-nine mgs. of radium were used for fifteen minutes at first, and then 44 mgs. Mr. Lawson thought few applications with a potent dose of radium was better than many applications with a weaker quantity. Mr. McKenzie Davidson discussed the case, pointing out the importance of being sure the strength of radium used was what it was said to be, as he had found serious discrepancies. It was also very important to cut off those radium emanations which were not needed in the cure.

Mr. R. R. CRUISE read a paper on the **Abuse of atropin in refraction work.** The paper was based on the examination of 140 eyes, in patients under sixteen years of age. He concluded that, on the whole, homatropin and cocain were quite equal, if not superior, to atropin, though in most cases the result after the two mydriatics was identical. The paper was discussed by Mr. STEPHENSON, Mr. BISHOP HARMAN, and Mr. DEVEREUX MARSHALL, the latest of whom alluded to Mr. LANG's investigations on the matter; and Mr. ERNEST CLARKE said it would have been useful to learn what the astigmatism was in the cases.

Mr. MAYOU read a communication on the **Disappearance of the iris from the pupillary area, following injury.**

REPORT [OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. J. R. SHANNON, Secretary.

MEETING OF MONDAY, MARCH 15, 1909. DR. WALTER E. LAMBERT, PRESIDENT, IN THE CHAIR.

Present, forty-two members.

Dr. E. Gruening presented a specimen of **Streptothrix masses from the upper canaliculus**.

This is a rare condition, there being only four on record. Over thirty years ago Dr. Gruening had reported a case. In this case, there were lachrymation, redness, and itching of the eye. A tiny drop of pus exuded from the upper canaliculus. Dr. Gruening slit the canaliculus and removed the masses. The cure was instantaneous.

Dr. C. J. Kipp reported a case of **Recurrent hemorrhage in the retina and vitreous and retinitis proliferans in a young man with surgical tuberculosis.**

This report is published elsewhere in this number.

Dr. E. Torok showed a case of **Tuberculosis of the conjunctiva treated with tuberculin.**

A girl two years old was brought to the hospital at the end of January. The mother noticed, three months ago, that the child's eyelids were swollen and red, and glued in the morning, discharging a considerable quantity of muco-pus. Simultaneously, a small, hard swelling appeared in front of the left ear. The mother was healthy; the father is suffering from pulmonary tuberculosis. Otherwise, these was no history of tuberculosis on either side. The child had never suffered from any illness except measles. The eyelids, particularly the

upper, were swollen. The palpebral fissure was smaller than normal. The tarsal conjunctiva on both sides, particularly on the upper lid, was infiltrated, uneven from granulations, scattered with yellowish red nodules. The bulbar conjunctiva somewhat injected. The cornea normal. There was much muco-purulent secretion. The preauricular gland showed fluctuation and subsequently suppurated. The clinical picture itself led us to the diagnosis of tuberculosis of the conjunctiva. Nevertheless to ascertain the correctness of the diagnosis, a piece of the conjunctiva was cut out. A small portion of it was inoculated into the anterior chamber of a guinea-pig,—the remainder was submitted to a pathological examination, which showed a typical tuberculous growth which can be seen under the microscope. In the eye of the guinea-pig a typical large conglomerate tubercle developed in the fourth week.

Tuberculosis of the conjunctiva is one of the less frequent eye diseases. Mules found one in thirty thousand, Spanger one in twenty thousand, Hirschberg one in six thousand eye patients. These data, however, are of no great significance because, with the advance of our knowledge of tuberculosis as an important etiological factor in eye diseases, the number of cases found increases from day to day. The infection may be ectogenic or endogenic. Experiments on animals have proven beyond doubt that endogenic infection exists, still these cases are less frequent than those of ectogenic origin. In the majority of cases of such affections, the patients are found, aside from the eye disease, in perfect health, and no other tuberculous focus can be found in their organisms.

Valude's experiments showed that the intact conjunctiva possesses a strong resisting power against tuberculous infections and that infection can only occur when there is a break in the epithelium. This injury of the epithelium may be caused by foreign bodies or by the rubbing of the eye with infected hands or handkerchiefs. The infection may extend to the conjunctiva through continuity from the face, lids, lachrymal sac, or nose. Tuberculosis of the conjunctiva is a disease of childhood and early life. It occurs most frequently in the first decade, less frequently after that, and very rarely after the fiftieth year. The clinical picture of the conjunctiva

is so variable that it is difficult to describe a typical case. Sattler was the one to classify the various clinical forms, and, to-day his grouping is accepted by most authors. It is as follows:

1st: This form as a rule is localized on the tarsal conjunctiva where ulcers of considerable size and depth appear, with sharply defined edges. Enlargement of the preauricular gland is seldom absent.

2d: In these cases, small nodules irregularly scattered are found. They are about the size of flaxseeds, grayish or grayish-yellow in color, and resemble trachoma. Later, the disease spreads to the cornea in the form of thin pannus. The preauricular gland is not always enlarged.

3d: In this form the eyelids become thick and the conjunctiva is covered with large exuberant granulations. The preauricular gland is generally enlarged.

4th: This form is lupus of the conjunctiva.

We seldom find cases which fit exactly into one of these groups. They occur usually as a mixed type. The prognosis with regard to the conjunctiva may be considered good, as the majority of cases can be cured. Naturally, the danger which may arise from the eventual generalization of the disease should not be neglected. As to therapy, authors are divided into two groups. Some advocate medical therapy and some surgical interference. In the present case, I advocate Koch's new tuberculin and shall have the pleasure of demonstrating the cases in due time after the termination of the treatment.

Dr. J. B. THOMAS presented two cases of **Sudden blindness from wood alcohol.**

The first, an Italian sixty-four years old, nine weeks before presenting himself, had become blind within a few hours. Both disks were decidedly pale and atrophic. Vision: light perception in each eye. He was accustomed to drink cognac and cordials in the morning. No other etiological factor was discovered The patient failed to return for further examination.

2d: J. F., a Polish Jew, fifty-four years old, a steady drinker of strong liquor. Five days before his appearance he drank hot "White Schnapps" in the evening and was almost totally blind the next morning. On examination, the

vision was $\frac{1}{200}$ in each eye. Pupils four to five millimetres, and sluggish. The only change in the fundi was dilatation of the veins and slight œdema of the disks. Urine negative. Treatment—pilocarpin gr. $\frac{1}{8}$ hypodermically daily and saline cathartics daily for ten days. In two days after treatment, vision right eye $\frac{3}{200}$, left eye unchanged. Ordered saturated solution of iodide of potassium 20 gr. t. i. d.

March 2d, about four weeks after the attack, vision in the right eye $\frac{14}{200}$. Pupils four millimetres, and react normally to light and convergence. Vision left eye $\frac{3}{200}$ and there is beginning pallor of the temporal edge of the disk.

March 13th: Vision right eye $\frac{20}{30}$; left eye $\frac{1}{200}$ excentrically. Both peripheral fields normal for white. Low central color vision in the right eye, and only slight for red in the left. Arteries of both fundi about normal size. Veins still dilated. Disk of the right eye slightly pale. The left distinctly pale and stippled.

In this connection, it is suggestive that General Wood has recently issued an order calling attention to the frequency of wood alcohol poisoning in the army, and warning officers and men of the Department of the East against it.

Dr. S. F. HALLOCK presented a case of **Persistent hyaloid artery, with unusual features.**

Dr. Hallock explained that the case was not certainly one of persistent hyaloid, but had been put down as that for want of a better characterization. With the ophthalmoscope, it showed very beautifully, stretching forward into the vitreous, and turning upon itself and twisting once or twice around the main stem. The vessel carried blood, and pulsated slightly.

Dr. E. F. KRUG for Dr. W. B. MARPLE reported a case of **Tuberculous iritis, showing the extraordinary effects of the injection of tuberculin.**

The report was accompanied by a series of beautiful illustrations by Dr. Young, showing the progress of the case towards recovery.

2d: a case showing a **BB shot on the floor of the orbit, previously localized.**

In this case, the foreign body had not been removed as it had given rise to no symptoms.

Dr. DAVID WEBSTER presented a patient with **Cyst of the iris.**

At the age of twelve years he had been struck in the eye, which was considerably injured. The eye had remained quiet until quite recently, the patient now being forty-eight years of age. Four weeks ago it became red and inflamed. He has a traumatic cataract and has perception of light in a small portion of the field. The cyst is readily seen by reflected light. Dr. Webster proposed to enucleate the eye.

Dr. J. WOLFF presented a patient **upon whom he had operated for high myopia.**

The patient had twenty dioptries of myopia which, when corrected, gave $\frac{20}{100}$ vision in each eye. There were central maculæ. Last summer he needled the left. One week subsequently he did linear extraction on account of the tension. The vision was now $\frac{1}{10}$ without a lens. The right eye was needled last January. The first needling had no effect. A week later a second was done, with very little reaction. Absorption was now going on slowly.

Dr. MARTIN COHEN presented a patient with **Tuberculous iritis.**

There were no other signs of the disease to be found. There were nodules on the pupillary margin of the left eye, and also at the ciliary border. It was a question whether the case was tuberculous or syphilitic, but he had concluded to use the tuberculin treatment.

In the discussion of Dr. Gruening's case, Dr. DAVID WEBSTER thought that the condition must be very rare, as he had never seen one in his long years of practice.

There was no discussion in Dr. Kipp's case.

In the discussion of Dr. Torok's and the other tuberculous cases, Dr. C. J. KIPP said that he had several cases of chorioretinitis, with deposits on Descemet's membrane. In one of these he had intended to use tuberculin, but the patient improved without it. He would undoubtedly, had he used the remedy, have put the recovery down to its credit.

In the discussion of Dr. Thomas's cases, Dr. E. GRUENING stated that he had seen ten cases this winter and had reported the matter to the Board of Health. One case was due to

Schnapps, one to Kummel, and one to Columbian spirits. The Commissioner of Health had asked for the names and addresses of these people, and is trying to find out where this material is obtained. The pathological condition is one of optic neuritis. The vessels are smaller, and the pupil large. When the disk is white, the vessel small, etc., the prognosis is bad; but when there is slight neuritis, prognosis is better. He has seen cases where there were no changes in the fundus. These are usually cases where the alcohol has been inhaled, as, for example, by workmen who have used shellac in brewery vats. He had seen two of these become blind, and the fundi were hyperæmic. They recovered. Another case was that of a child which was brought to the hospital with both pupils dilated, and apparently blind. The picture was complete. Alcohol baths had been given in a dark, unventilated room for some febrile condition. Wood alcohol is used in a number of substances where its presence is unsuspected, for instance, essence of peppermint. Dr. W. E. LAMBERT agreed with Dr. Gruening that these cases should be reported to the Health Commissioner. Dr. H. H. TYSON had seen four cases in the last two months. Wood alcohol and caramel, called coffee, obtained at Italian saloons, was the cause. There is usually in these cases deterioration of vision, subsequent to improvement. Dr. C. W. CUTLER had seen a case a year ago where the man had bathed a broken arm in wood alcohol. The picture was typical. There was double optic atrophy. Dr. C. J. KIPP thinks that these cases are not as numerous as is apparent. They go from one clinic to another, and many of us see the same cases.

In the discussion of Dr. Hallock's case, Dr. J. H. CLAIBORNE said that, in 1898, he had reported a similar case, and had looked up the literature on the subject very thoroughly. Dr. E. GRUENING said that some time ago Dr. Schapringer had shown a similar case. Dr. Gruening thought this was a vein, undoubtedly. Dr. C. J. KIPP said that he considered such cases branches of the retinal artery, turning on themselves and rejoining the circulation. Hyaloids are a brownish color almost always. These he described years ago.

In the discussion of Dr. Webster's case, Dr. E. S. THOMSON said that it was a very striking case. He had never seen one

quite like it. Implantation cysts had been well described by Treacher Collins and Oatman. Dr. E. GRUENING did not think this an implantation cyst. Dr. Webster said that he would enucleate the eye: 1st, because it is useless; 2d, because it is inflamed and painful; and 3d, because, if merely the cyst itself were removed, it would probably recur.

In the discussion of Dr. Wolff's cases, Dr. LINN EMERSON said that he had done a double extraction a few years ago, which had had a very curious denouement. He removed one lens with an excellent result. He then operated upon the second. The patient was compelled to wear glasses for her close work, which she protested against vigorously. Dr. W. E. LAMBERT had had four very satisfactory cases which he had previously reported.

SECTION MEETING, MONDAY, APRIL 19, 1909. DR. W. E. LAMBERT, PRESIDENT, IN THE CHAIR.

Present, thirty members.

Dr. DAVID WEBSTER presented a patient from whose eye had been removed a **cyst of the iris** one-quarter of a century ago. After the operation a cataract developed, which was extracted, with a resulting vision of $\frac{20}{50}$. Vision remains the same to-day.

Dr. A. E. DAVIS presented a patient with **sarcoma of the choroid and ciliary body with retinal detachment.**

Dr. W. E. LAMBERT presented a patient upon whom he had **operated for ectropion** by means of a Wolfe graft. The condition was the result of an attack of erysipelas.

Dr. E. M. ALGER showed a patient with **embolism of the inferior retinal artery.** The patient was a boy of nineteen, in good health. No alcoholic or venereal history. He had been taking no drugs of any kind. The attack of blindness in the eye came on after spending the preceding evening at reading and while washing the face. After a time the sight returned slightly, but a cloud remained. Ophthalmoscopic examination showed complete obliteration of a portion of the inferior temporal artery at the periphery of the fundus, beginning just above and including a bifurcation of the vessel, which is absolutely white in color. Both branches

below the obstruction contain blood, but the column is interrupted. Vision $\frac{20}{30}$ without glasses and Jaeger I.

Dr. HENRY W. WANDLESS made a preliminary report on several cases of blind idiots in one family to which he improperly gave the name **Amaurotic family idiocy.** This condition, as Dr. Wandless pointed out, is rarely found except among the Jewish people. His cases were not Hebrews, but in a family known to be five-sixths Irish. Both father and mother were well and hearty. There is nothing in the family history to point to hereditary causation. The first case died at the age of fourteen. The visual defect was first noticed at the age of seven. Shortly after, he suffered failing mentality, and for some time before death was bedridden and extremely emaciated. The second case is a boy now twelve years of age. He was well until his eighth year, when his eyes began to fail, and fifteen months later mental symptoms developed. The third case, a girl, eight years of age, is the sixth child of the family. She at the present time shows a slight hyperæmia of the choroid and retina, and considerable irritability of temper.

The second case, Frank, presents the following eye conditions: External appearance of the eyes, normal. The pupils are dilated, but contract to strong daylight. The transparent media are normal. Vision amounts to seeing objects imperfectly. The eyes keep up a constant, irregular movement. At the first examination, the retina in both eyes was œdematous, the choroid congested, and the optic nerves atrophic. Since then the œdema of the retina has disappeared, and atrophy of the choroid has begun. The characteristic changes in the macular region are not present, and there is nothing to indicate that they ever existed.

Dr. Brooks's report of the gross findings at the autopsy in the first case were added. He reports that the brain was very small, being reduced to about the size of that of a twelve months' infant, and its weight, including the cerebellum and medulla, was twenty ounces. The cortex is of a peculiar, yellowish color. The convolutions are very small, more like wrinkles over the surface. The consistency of the brain tissue is abnormally firm. The frontal lobes are extremely atrophied, the chief decrease in size appearing to be from shrinkage of the white matter.

Dr. GEORGE YOUNG made a report on **Mr. Sutcliffe's keratometer.** He explained that his investigation of the instrument was for the purpose of testing its reliability as compared with the Javal ophthalmometer. Fifty-six eyes were tested. Astigmatism of as many varieties and degrees as available were chosen. The eyes were first tested with the keratometer; second, with Javal's ophthalmometer; third, by retinoscopy; fourth, by direct ophthalmoscopy, and fifth, by the test types. The average difference in dioptries of astigmatism between the two instruments was 0.205 D. The average difference in the degree of the axis was 2.05. It was stated that the instrument is new in principle, and reliable. Stress was laid on the following points:

1. The axes are read in one position, without rotation or movement;
2. The technique is reduced to three moves in all cases alike;
3. The focus is easier;
4. The instrument is self-reading. There is no counting or estimating of steps or fractions thereof;
5. There is only one mire, and that is movable;
6. It is particularly valuable in restless patients and unsteady eyes;
7. It saves time.

The conclusions were that the instrument was worthy of consideration, and that there were no apparent disadvantages that could not easily be remedied.

Dr. ALEXANDER DUANE read a paper entitled the **Prognosis in eye diseases, considered from the standpoint of the patient's rights and the ophthalmologist's duty.**

Dr. Duane began by saying that perhaps a better title for his paper would be: "What ought we to tell a patient afflicted with a serious and progressive eye disease?" The important thing to the patient is the prognosis in his case, and too often this is given hastily and without thought of the effect that may be produced on his mind. The result is untold mental suffering, leading sometimes to despair—even to insanity or suicide—or more often driving the patient to resort to quacks, whose business it is to promise hope to the hopeless. Thus arises frequently a contempt for the

ophthalmologist and the science that he represents. The author thought it scarcely ever justifiable to tell a patient that he cannot recover or that nothing can be done for him. This sort of statement is oftenest made in:

1. Extensive disease of the cornea, dense corneal opacities, staphyloma, keratoconus;
2. Irido-cyclitis, degenerative irido-choroiditis, and other degenerative or destructive affections of the uveal tract;
3. Retinitis pigmentosa;
4. Optic nerve atrophy (including high-grade toxic amblyopia);
5. Glaucoma;
6. Progressive high myopia;
7. Detachment of the retina;
8. Intraocular hemorrhage;
9. Cataract;
10. Injuries, including operations.

Dr. Duane then detailed several cases illustrating the folly—and very often the cruelty—of giving a hopeless prognosis to the patient. He dwelt especially upon the uselessness and wrongfulness of telling patients with slight opacities in the lens that they had "cataract," and concluded with the statement of a number of propositions which he considered justified. These emphasized the value, both to patient and surgeon, of a cheerful optimism in almost every variety of ophthalmic affliction. "We know not how soon some new discovery may change our views as to the diagnosis or prognosis in a given case. Only recently we have learned that sometimes an apparently intractable uveal disease may be relieved by tuberculin; optic neuritis that we once would have considered irremediable may be relieved by decompression; an optic nerve atrophy that we would have held to be inevitably progressive may be arrested by operation on the accessory sinuses, and an impending disastrous suppuration in the eye may be checked by staphylococcus vaccine." Brutal positiveness was denounced as sometimes simply a mask of ignorance, the expression of a hastily formed and erroneous opinion; not forgetting, however, that such frankness must not be withheld when its expression is necessary—as, for example, in a case of sympathetic inflammation.

Finally, Dr. Duane said: "To the patient our prognosis should be as encouraging as possible, our demeanor sympathetic and cheerful, our treatment such as will best relieve his symptoms, so that if we cannot cure or even arrest his trouble, we shall at least make him the better for having seen us. In this way we shall best subserve the patient's interests and perform our duty as ophthalmologists and physicians."

The paper was sympathetically discussed and cordially approved by Drs. Marple, Graef, Pooley, Gruening, Claiborne, Webster, Davis, and Lambert.

Dr. Webster's case was discussed by Dr. T. R. POOLEY.

In the discussion of Dr. Davis's case, Dr. E. GRUENING pointed out that detachment of the retina is rare in sarcoma of the ciliary body. Dr. Davis explained that transillumination was practically as good at the site of the growth as in other positions. He used a Wuerdemann transilluminator. Dr. W. B. MARPLE could not understand how this could be. The case was also discussed by Dr. DAVID WEBSTER and Dr. E. B. COBURN, Dr. COBURN pointing out that some flat tumors were capable of almost perfect transillumination.

In the discussion of Dr. Lambert's case Dr. T. R. POOLEY mentioned that he had described an operation for ectropion in the *Transactions of the American Ophthalmological Society* some years ago. The case was also discussed by Drs. E. GRUENING and W. B. MARPLE.

There was no discussion of Dr. Alger's case.

In the discussion of Dr. Wandless's report, Dr. E. GRUENING said that the characteristic changes were not present in these cases, and that the ophthalmoscopic findings described by Tay are of a different type. These cases of Dr. Wandless were not cases of amaurotic family idiocy. They had been described previously by German writers, and came in older children.

Dr. Young's report on the Sutcliffe keratometer was discussed by Dr. FRANCIS VALK and Dr. A. E. DAVIS.

The Chairman announced that the matter of advising the President to call an International Congress on Ophthalmia Neonatorum having been left in his hands for decision, he had concluded that no action be taken by the Section.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE FIRST AND SECOND QUARTERS OF THE YEAR 1908.

By Dr. G. ABELSDORFF, in Berlin; Prof. ST. BERNHEIMER, in Innsbruck; Dr. O. BRECHT, Prof. R. GREEFF, Prof. C. HORSTMANN, and Dr. R. SCHWEIGGER, in Berlin; with the assistance of Prof. A. ALLING, New Haven; Prof. E. BERGER, Paris; Prof. CIRINCIONE, Genoa; Dr. DALÉN, Stockholm; Prof. HIRSCHMANN, Charcow; Dr. J. JITTA Amsterdam; Mr. C. DEVEREUX MARSHALL, London; Dr. H. MEYER, Brandenburg; Dr. P. VON MITTELSTÄDT, Metz; Dr. H. SCHULZ, Berlin; Prof. DA GAMA PINTO, Lisbon; and others

Translated by Dr. PERCY FRIDENBERG, New York.

Sections IV.–VII. Reviewed by Dr. ABELSDORFF.

IV.—ANATOMY.

53. v. SZILY, A. **Development of fibrillary supporting fibres in the embryo and their relation to the vitreous.** *Anatom. Hefte*, 107, p. 651.

54. LANDMANN, O. **Microphthalmos in a chick embryo eight days old.** *Centralbl. f. prakt. Augenheilkunde*, xxxii., p. 131.

55. LANGE, O. **Explanation of the arrangement of retinal layers in vertebrate and non-vertebrate eyes.** *Centralbl. f. prakt. Augenheilkunde*, xlvi., No. 1, p. 633

56. BARTELS. **Primitive fibrils in the axis-cylinder of the optic nerve and the interpretation of varicose axis-cylinders.** *Arch. f. Augenheilkunde*, lix., No. 2, p. 168.

57. LAUBER, H. **Contribution to embryology and comparative anatomy of iris and retinal pigment epithelium.** *Arch. f. Ophth.*, lxviii., 1, 1.

58. WOLFRUM. **Musculature of the chromatophores of the iris** *Ophth. Klinik*, 1908, No. 7, p. 193.

59. GRADON, J. T. **Development of the lens.** *Ophthalmoscope*, Sept., 1906; March, 1907.

v. SZILY (53, **Development of fibrillary supporting fibres**

in the embryo and their relation to the vitreous) finds that cells of the mesenchyma and fibrillary interstitial tissue form the components of embryonic connective tissue. The vitreous is a part of the general embryonic supporting tissue which has been differentiated, its first rudiment being formed by an intercellular bridge leading from a cell of the primitive retina to its corresponding cell in the primitive lens. The lens is finally excluded by the development of the non-vascular lens capsule.

LANDMANN (54, **Microphthalmos in a chick embryo eight days old**) found the right side of the head fully developed while the left was rudimentary. This lack of development of the left side of the brain was the cause of the microphthalmus.

LANGE (55, **Explanation of the arrangement of retinal layers in vertebrate and non-vertebrate eyes**) notes that the ocular rudiment in vertebrates arising from the medullary groove becomes displaced into the interior of the encephalon and does not come into relation with the ectoderm until a later period, as the primary optic vesicle, while the eye of the invertebrates arises directly from the epidermis at a distance from the medullary groove. This explains the distal position of the optic ganglion and the proximal position, away from the light, of the light perceptive layers of the retina in vertebrates.

BARTELS (56, **Primitive fibrils in the axis-cylinder of the optic nerve and the interpretation of varicose axis-cylinders**) describes the optic-nerve fibre as composed of:

1. A very thin medullary sheath.
2. The axis fibrils.
3. The axoplasma, a perifibrillary substance.
4. The myelaxostroma, in connection with the medullary sheath.
5. The fibrillary acid, which probably clings to the axis fibrils.

Varicosities may be caused as artefacts in fixation, in which case the swollen portions represent the normal part of the fibrils while the narrow portions are shrunken. Actually pathological varicosities, as observed in tissue œdematous with lymph, are, on the other hand, decidedly thicker than

normal cylinders and represent a swelling probably due to imbibition.

LAUBER (57, **Contribution to embryology and comparative anatomy of iris and retinal pigment epithelium**) finds that the first traces of retinal pigment in man appear at the equator of the inner portion of the external layer of the primitive optic vesicle. The pigment spreads forward more rapidly than toward the posterior pole. The retinal pigment layer of the iris reaches the ciliary body about the seventh month. Pigmentation of the stroma cells was never seen until the ninth month. The development of the iris is not complete at birth as shown by the slight action of atropin and is not definitely finished until the second or third year.

WOLFRUM (58, **Musculature of the chromatophores of the iris**) claims that Muench has not proven the existence of myofibrils in the protoplasm of the chromatophores of the uveal tract. The alleged muscle-cells show double refraction like all contractile substance. The longitudinal striation of the chromatophores is common to the protoplasm of all cells and does not prove their muscular nature.

GRADON (59, **Development of the lens**) finds that during the early stages of development there is a flattening of the anterior surface of the lens in those animal eyes which when at rest are accommodated for distance, while in eyes at rest in accommodation for short range, as in the trout and shark, there is no such flattening. The lymph space which develops about the equator may be closed by excessive accommodation effort, particularly in hypermetropia, and thus interfere with the nutrition of the equatorial lens fibres. This may play a rôle in the etiology of senile cataract which always begins at the equator and is associated with hypermetropia or astigmatism in three quarters of the cases.

V.—PHYSIOLOGY.

60. HESSE, R. *Vision of lower animals.* Jena, G. Fischer, 1908.

61. URBANTSCHITSCH, V. *Subjective acoustic phenomena and subjective visual concepts.* Leipzig u. Wien, Deuticke, 1908.

62. GRUETZNER, P. **Localization of trans-scleral illumination.** *Pflueger's Arch. f. d. ges. Physiol.*, No. 121, p. 298.

63. BRENNER, E. **Modification of Hering's theory of complementaries.** *Ibid.*, No. 121, p. 370.

64. v. TSCHERMAK. **Simultaneous contrast in various sense organs.** *Ibid.*, No. 122, p. 89.

65. STIGLER, R. **Discrimination threshold in the rising portion of a light stimulus.** *Ibid.*, No. 123, p. 163.

66. STIGLER, R. **Flickering of cinematograph pictures.** *Ibid.*, No. 123, p. 224.

67. GUTTMANN, A. **Studies in color weakness.** *Zeitschrift f. Sinnesphysiol.*, No. 42, pp. 24 and 250.

68. BOSWELL, F. P. **Necessary stimuli of visual organ in the fovea.** *Ibid.*, p. 299

69. LANGFELD, H. S. **Light sensitiveness and pupillary width.** *Ibid.*, p. 349.

70. BOLTUNOW, A. **Visual acuteness in colored light.** *Ibid.*, p. 359.

71. CORDEIRO, F. **Color sensation.** *Ibid.*, p. 409.

72. RUPPERT, L. **Comparison of distinction capacity and perception of motion in the periphery of the retina.** *Ibid.*, No. 42, p. 409.

73. KOENIG, B. **Retinal function in vision.** *Ibid.*, p. 424.

74. STILFVAST. **Acuteness of vision for various colors at the centre of the retina.** *Skandinav. Arch. f. Physiol.*, xx., Nos. 5 and 6, p. 411.

75. GERTZ, H. **An experiment in direct vision.** *Ibid.*, p. 357.

76. HESS, C. **Studies of the vision and pupillary reaction of day-birds and of nocturnal birds.** *Arch. f. Augenheilkunde*, lix., No. 2, p. 143.

77. RISSLING, P. **Physiological variations in osmotic pressure of serum and of intraocular fluids.** *Ibid.*, lix., No. 3, p. 239.

78. ZEEMANN. **Form of the posterior surface of the lens.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 83.

79. RAEHLMANN, E. **Simultaneous color contrast.** *Zeitschrift f. Augenheilkunde*, xix., No. 1, p. 1.

80. ROCHE, CHAS. **Simple and rapid method of determining the presence of binocular vision.** *Ann. d'ocul.*, cxxxix., p. 42.

81. TSCHERNING. **Entoptic phenomena of retinal circulation.** *Acad. de méd. de Paris*, Mai 28, 1907.

82. ANGELUCCI, A. **Paintings by Daltonists.** *Recueil d'opht.*, xxx., p. 1.

83. CONSIGLIO, A. **Observations on the entrance of fluorescin into the eye.** *Revista Ital. di Ottalm.*, iv., Nos. 1–2, 1908.

HESSE (60, **Vision of lower animals**) notes that pigment is not essential for the stimulation of visual cells. Pigment represents but one form of optical differentiation, the other being that by lenses. This produces functions of perception of distance, direction, and form in visual cells capable of stimulation by light and dark.

URBANTSCHITSCH (61, **Subjective acoustic phenomena and subjective visual concepts**) deals with sensations of sound and

color which accompany unconscious cerebration. Various color sensations may modify the subjective perception of musical compositions. Simultaneous excitation of the two eyes by two different colors produces changes in the subjective hearing which do not occur with one.

GRUETZNER (62, **Localization of trans-scleral illumination**) notes that in transillumination of the nasal half of the sclera the sensation of light is referred to the temporal side, while a similar localization to the temporal side occurs when the transillumination takes place on the temporal side. This is not contradictory to the accepted theories of retinal impressions and is explained by the fact that there are no perceptive elements on the temporal side so far forward, so that the light is perceived only by the elements on the nasal side. Pressure phosphenes may be elicited within *7mm* of the corneal margin on the nasal side, and *12mm* on the temporal.

BRENNER (63, **Modification of Hering's theory of complementaries**) accepts, on the basis of the theory of chemical equivalents, a single reversible photo-chemical process in each of the three visual substances, which, according to its direction and speed, determines the visual sensation.

V. TSCHERMAK (64, **Simultaneous contrast in various sense organs**) notes that this phenomenon is by no means limited to the organ of vision, but can be studied with advantage as it affects the sense of motion, taste, touch, and temperature.

STIGLER (65, **Discrimination threshold in the rising portion of a light stimulus**) used as the threshold of differentiation, as to time, the smallest difference between two exposures during which one and the same light stimulus would produce a difference of apparent illumination. This was found to be proportional to the intensity of the stimulus.

According to STIGLER (66, **Flickering of cinematograph pictures**), this flickering is due to the successive dark intervals caused by change of film. Its recognition depends on persistence of the preceding light sensation. Shortening of the time of the separate light stimuli by revolving spokes, which break up the single long stimulus into a number of short ones and at the same time reduce the apparent luminosity, increases the time of primary light sensation so that the dark pause is bridged over, and the flickering disappears.

GUTTMANN (67, **Studies in color weakness**) divides the anomalous tri-chromates into red-weak and green-weak, as the threshold of perception for color is raised. Differentiation is less acute, and the time required for recognition of color is greater, than normal.

BOSWELL (68, **Necessary stimuli of visual organ in the fovea**) finds that the periphery of the retina in adaptation to darkness is much more sensitive to small stimuli than the region of the fovea.

LANGFELD (69, **Light sensitiveness and pupillary width**) was unable to explain the unusual size of the pupil by variations in sensitiveness, as this was noted in the dark and shows by instantaneous photographs.

BOLTUNOW (70, **Visual acuteness in colored light**) found that perception of red depends only slightly on the illumination, whereas acuteness of vision for green diminishes rapidly with lessened illumination.

CORDEIRO (71, **Color sensation**) considers the plates of the outer members of the cones to be elastic bodies with special vibration periods, capable of taking up light vibrations of a similar period or its multiples.

RUPPERT (72, **Comparison of distinction capacity and perception of motion in the periphery of the retina**) determined the visual acuteness and the sense of motion at various points of a horizontal meridian running through the fovea. Vision falls slowly to a point 35° from the fovea, and then diminishes rapidly, while perception of motion falls but very little up to a point 55° distant. The recognition of motion is probably a special sense which does not depend on the same anatomical and physiological factors as visual differentiation.

KOENIG (73, **Retinal function in vision**) accepts Alefeld's biophysical hypothesis that colloid substances are positively phototropic. Visual purple is probably a colloid solution which tends to wander toward light and thus produces pressure on the rods and cones, producing sensations of light which are actually a form of tactile sensations.

STILFVAST (74, **Acuteness of vision for various colors at the centre of the retina**) determined the largest number of distinct light impressions which could be perceived on a given area of the fovea. He found that acuteness of vision for red

and green light was equal to that for white, while vision for blue was somewhat less.

GERTZ (75, **An experiment in direct vision**) determined the breadth of the angle of direct vision and of aberration of gaze. A given point under fixation may fall at any part of this field owing to uncontrollable slight motion of the eye. The area of direct vision and that of aberration have the same dimensions, viz., between 3′ 20″, and 4′ 5″.

HESS (76, **Studies of the vision and pupillary reaction of day-birds and of nocturnal birds**) tested falcons and buzzards with bits of white meat and found, as in doves and hens, that after adaptation to bright light, the violet end of the spectrum appeared shortened. In day-birds red-yellow and green rays have the most marked influence on the pupil; in night-birds, yellow, green-yellow, and green.

RISSLING (77, **Physiological variations in osmotic pressure of serum and of intraocular fluids**) determined the osmotic pressure of the aqueous and vitreous in various domestic animals and found that it may be equal to or greater or less than that of the serum.

ZEEMAN (78, **Form of the posterior surface of the lens**) followed up v. Pflug's observations of a physiological lenticonus posterior during accommodation, as shown by fixation in liquid carbonic acid. In two cases examined under mydriasis he noted doubling of the posterior lens image (Purkinje) during accommodation. This is explained by the fact that the posterior surface of the lens acts as a concave mirror near the centre and as a convex mirror at the periphery, producing two images.

RAEHLMANN (79, **Simultaneous color contrast**) opposes the view expressed by Heine and Lenz that average normal color vision shows only slight individual variations. The method of determining the threshold for contrasts by means of colored papers he considers defective.

ROCHE (80, **Simple and rapid method of determining the presence of binocular vision**) has the patient rapidly touch a small spherical object, situated at a distance of *25cm* with the point of a knife. This is done promptly if binocular vision is present, but fails if there is none.

TSCHERNING (81, **Entoptic phenomena of retinal circula-**

tion) finds the light of the mercury lamp better than the sky as a background. The white blood cells probably act as minute lenses concentrating the rays on the light-perceiving layer behind the vessels. Tscherning concludes that as blue light shows the circulation better, the blue-perceiving layer must be nearer the vessels than the layer for red and green. BERGER.

ANGELUCCI (82, **Paintings by Daltonists**) has collected art works by red-green color-blind painters, and found that light effects were represented by red, shadows by green (Liebreich's symptom). Practice enables the color-blind to distinguish the various tones of red, but not those of green. Violet is used for shadows by the red-green blind and for the parts in light, as well by the totally color-blind. Exaggeration of the light effects, representing green as yellow green, or blue green, according as it is in bright or medium light, or in shadow, and excessive polychromy, representing brown as yellow, as red, and as violet or green under the same conditions of illumination, are characteristic mannerisms. BERGER.

CONSIGLIO (83, **Observations on the entrance of fluorescin into the eye**) found that a minimum dose of *3ccm* to the kilo of body weight was necessary in dogs to produce Ehrlich's line which is an index of the amount of fluorescin in the circulation. This line may vary but seems to depend on gravity and diffusion, as it may be made to change its direction by altering the position of the head post mortem. Warmth increases, and changes in the composition of the aqueous alter, the rate of diffusion of fluorescin from the blood into the intraocular fluids. CALDERARO.

VI.—REFRACTION AND ACCOMMODATION.

84. THORNER, W. **Relation between near work and myopia.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 10.

85. THORNER, W. **Significance of near work in development of myopia.** *Ibid.*, p. 174

86. MUEGGE, F. **Frequency of astigmatism and relation to vision.** *Ibid.*, p. 474.

87. STILLING, J., and LANDOLT, H. **Relation of light sense to refraction.** *Ibid.*, p. 490.

88. STOCK, W. **Cavernous optic atrophy in myopia.** *Ibid.*, p. 342.

89. Fridenberg, Percy. **Over-correction of hypermetropia in treatment of asthenopia.** *Ibid.*, p. 21.
90. Behse, F. **Anatomical structure of myopic crescent.** *Arch. f. Ophth.*, lxvii., No. 3, p. 379.
91. Schulin, C. **Real cause of myopia.** *Wochenschr. f. Hyg. u. Ther. d. Aug.*, xi., No. 15, p. 115.
92. Katz, K. A. **Ophthalmic migraine in ametropia.** *Russky Wratsch*, 1908, No. 6.
93. Amar, J. **Effect of sun-rays on sight.** *Journ. de phys. et path. gén.*, March 2, 1908.
94. Plantenga. **Spasm of accommodation.** *Ned. Tyds. v. Geneesk.*, No. 1, p. 10, 1908.
95. Wentink. *Contribution to study of myopia.* Inaug. Dissert., Amsterdam, 1908.
96. Percival, A. **Some prismatic effects of lenses.** *Ophthalmoscope*, Jan., 1908.
97. Cooper, J. **Simple notes on oblique prisms.** *Ophth. Review*, March, 1908.

Thorner (84, 85, **Relation between near work and myopia**) notes as many as seven interrupted motions of the globe per second in reading, somewhat less frequent in drawing and writing, and almost continuously in needle work. This explains why typesetting induces myopia more easily than watch-making or sewing. The nearer the reading matter is brought to the eye, the larger and more rapid the excursions.

Muegge (86, **Frequency of astigmatism and relation to vision**) found 10.5% of astigmatics in 15,385 eye patients, 47% myopic, 40% hypermetropic, and 10% mixed, indicating astigmatism as a factor in myopia. Degrees of 0.5–1 D. are most common, and more common in myopic than in hypermetropic astigmatism. Mixed astigmatism is generally above 3. D. Vision] is better in myopic than in hypermetropic astigmatism.

Stilling and Landolt (87, **Relation of light sense to refraction**) tested 236 cases of myopia and 100 of hypermetropia. In the light, differentiation and sensitiveness to light were up to the normal. These functions were affected only in case of choroidal or retinal disease. Light sensation in twilight was diminished only by retinal lesions or spasm of accommodation.

Stock (88, **Cavernous optic atrophy in myopia**) describes five globes in which lacunæ were found. The ophthalmo-

scopic picture has been seen only in glaucoma and myopia. In both, the process is due to traction on the nerve fibres by pressure or tension, causing them to break off at the lamina, leaving cavities. This may explain the unexpectedly poor sight of some myopes whose macular region shows no changes.

Fridenberg (89 **Over-correction of hypermetropia in treatment of asthenopia**) notes that low degrees of ametropia are most apt to cause eye-strain by inducing spasm of accommodation, which is not so apt to develop in high degrees accompanied by visual defect. Atropin may act well for a time, but the ciliary spasm recurs. Over-correction by about 1 D. somewhat fogging the vision for distance permanently relaxes the accommodation, and may be diminished or cancelled fully, later.

Behse (90, **Anatomical structure of myopic crescent**) lays stress on a choroidal circumscribed atrophy and loss of pigment due to traction. Distraction crescent (Stilling) and implication of a fold of the optic nerve (Heine) are unusual. Atrophic patches may depend on tears of the lamina elastica and appear as white spots with a border of pigment or as small pigment deposits. Deep retinal deposits with pigment invasion cannot be differentiated from choroidal patches.

Schulin (91, **Real cause of myopia**) considers scrofula "the mother of myopia." Toxins are formed in enlarged lymph glands which enter the eye through the circulation and cause refraction error or even squint.

Katz (92, **Ophthalmic migraine in ametropia**) reports three cases cured by correction of hypermetropia and hypermetropic astigmatism.

Amar (93, **Effect of sun-rays on sight**) notes an unusually high percentage of astigmatism against the rule in sunny countries, notably Southern Italy, Tunis, Tripoli, Algiers, and Morocco, appearing in from 12% to over 50% of the population. This is explained by ciliary spasm communicated to the sclera by the insertion of the muscle of accommodation.

Plantenga (94, **Spasm of accommodation**) considers clonic spasm of no importance and has found true tonic spasm of accommodation only once in examining 1000 cases.

Wentink (95, **Contribution to study of myopia**) discusses the etiological factors, progress, and treatment. Maculæ, regular

astigmatism, and congenital cataract increase axial myopia. Widmark's hypothesis that marked maculæ in one eye are always associated with greater ametropia on that side does not hold good. Myopia may progress at any age, but is most common under 16, and advances more frequently and rapidly in high degrees. Total correction has a good effect on this feature, is valuable on account of the improvement in central vision, and diminishes the insufficiency of convergence. Poor accommodation and subjective discomfort are the only counter indications to total correction.

PERCIVAL (96, **Some prismatic effects of lenses**) points out that often patients who require a different correcting glass for each eye complain of discomfort when reading. This is due to the fact that when the patient looks down the prismatic effect is greater in the stronger lens. For example if one lens be + 3 D. and the other + 5 D. there is a difference of 1° 23′ in the angle the eyes have to drop. This cannot be supported for long. To obviate this the strong glass should be decentred 5.5*mm* down. This question is of great importance when bifocals are ordered, for by suitably decentring the optical centre of the reading glass, the real centres of the combination can be brought into the correct position.

T. HARRISON BUTLER.

COOPER (97, **Simple notes on oblique prisms**). The subject of oblique prisms is treated under three heads. (1) Horizontal and vertical components of an oblique prism. (2) To determine the numerical value in degrees of any prism placed obliquely in the trial frame. (3) The simultaneous correction of horizontal and vertical deviations by a single prism. The results obtained are: (1) The vertical rectangular component of an oblique prism is that prism which, placed at 90°, is equal in vertical deviating power to the given oblique prism, and is equal to the sine of the angle which the latter makes with the horizontal multiplied by its value in degrees.

The ratio of the vertical to the horizontal prism measures the tangent of the angle which the resultant prism makes with the horizontal.

The horizontal equivalent of an oblique prism is that prism which, placed at 180°, is equivalent in horizontal deviating power to the given oblique prism, and is equal to sin (90°—

angle which the latter makes with the horizontal) multiplied by its value in degrees.

The resultant of any two prisms which are rectangular components is equal to the square root of the algebraic sum of the squares of the prisms.

The method is really easy with the aid of a table of natural sines and tangents and one of four-place logarithms which Burdon Cooper says "are to hand in most consulting rooms." We can, however, thank him for one small mercy, for "a table of square roots is unnecessary." T. HARRISON BUTLER.

VII.—MUSCLES AND NERVES.

98. REDLICH, E. **Hysterical pupillary paralysis.** *Deutsche med. Wochenschr.*, 1908, No. 8, p. 313.

99. BECKER, F. **Amblyopia of strabismic eyes.** *Ibid.*, No. 7, p. 280.

100. KOELLNER. **Ætiology of abducens paralysis.** *Ibid.*, p. 112.

101. PEYSER, A. **Isolated abducens paralysis in otitis media.** *Berl. klin. Wochenschr.*, 1908, No. 28.

102. GOETERMANN, C. **Abducens paralysis after lumbar anæsthesia.** *Ibid.*, No. 33, p. 552.

103. LACHMUND, H. **Study of the convergence reaction.** *Ibid.*, p. 1263.

104. LEVINSOHN, G. **Meiosis in reflex pupillary paralysis.** *Ibid.*, No. 15, p. 745.

105. ABELSDORFF, G. **Unilateral reflex pupillary paralysis in oculomotor palsy.** *Med. Klinik*, 1908, No. 9.

106. STEINERT, H. **Oculomotor disturbances as focal symptoms of cerebral disease.** *Ibid.*, No. 25, p. 938.

107. HESS, C. **Physiology and pathology of the pupil.** *Arch. f. Augenheilkunde*, lx., No. 4, p. 327.

108. KRUSIUS, F. **Divergent strabismus in hypermetropia and the effect of mydriasis.** *Ibid.*, lx., Nos. 2 and 3, p. 272.

109. LEVI, E. **Voluntary squint in primary position of fellow eye.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 167.

110. BARTELS, M. **Simple phorometer for latent divergence.** *Zeitschrift f. Augenheilkunde*, xix., No. 2, p. 101.

111. OHM, J. **Retinal meridians after squint operation.** *Arch. f. Ophth.*, lxvii., No. 3, p. 439.

112. CASSIRER and LOESER. **Influence of rotation on nystagmus.** *Neurol. Centralb.*, 1908, No. 6.

113. POULAIN, G. **Colored glasses in treatment of intermittent squint.** *Rec. d'opht.*, xxx., p. 161.

114. GALEZOWSKI, J. **Congenital cicatricial retraction of levator and superior rectus with immobility of the globe.** *Soc. d'opht. de Paris*, June 2, 1908.

115. Bourdeaux, B. **Strabismus, functional and organic.** *Ibid.*, March 10, 1908.

116. Drouin. **Congenital amblyopia with divergent squint.** *Ibid.*, April 7, 1908.

117. Hedon, E. **Post-operative oculomotor paralysis in otitis media.** *Arch. internat. de laryngol.*, 1908, No. 2.

118. Landolt, E. **Clinical results in muscle advancement.** *Arch. d'opht.*, xxviii., p. 257.

119. Raymond, F., and Claude, H. **Tumor of hypophysis, paralysis of associated movements, anæsthesia of cornea.** *Soc. de neurol. de Paris*, Feb. 6, 1908.

120. Wyler, J. **Fleeting paralysis of ocular muscles.** *Arch. f. Ophth.*, xxxvii., No. 1, p. 16.

121. Culbertson, L. **Paralysis of ocular conjugate superduction.** *Amer. Journ. of Ophth.*, May, 1908.

122. Posey, W. C. **Ocular palsy in Graves' disease.** *Ophth. Record*, June, 1908.

123. Veasey, C. **Rhythmic alteration of palpebral fissure.** *Med. Journ.*, Jan. 18, 1908.

124. Roenne, H. **Disturbances of associated movements.** *Hospitalstid.*, 1908, p. 193.

125. Stephenson, S. **Associated movements of upper eyelid and jaw.** *Ophthalmoscope*, Jan., 1908.

Redlich (98, **Hysterical pupillary paralysis**) notes marked mydriasis with lack of reaction to light during hysterical or epileptic attacks. This depends on sympathetic irritation (?) as does the same pupillary condition noted during muscular exertion, which in normal subjects is associated with brisk contraction.

Becker (99, **Amblyopia of strabismic eyes**) finds defective vision as an antecedent in many cases of squint, which in turn increases the visual defect. In long-standing squint, correction may not aid vision, but after loss of the good eye vision may be increased by practice. Early treatment of squint by correction of ametropia and atropinization of the better eye is of importance.

Koellner (100, **Ætiology of abducens paralysis**) noted disease of the circulatory system, especially of the area of the internal carotid, in a number of cases.

Peyser (101, **Isolated abducens paralysis in otitis media**) reports a case of subperiosteal abscess opened with relief of abducens paralysis, and one of operative lateral sinus thrombosis followed by choked disk and crossed abducens paralysis.

Gradenigo's theory that the triad, otitis media, pressure pain and swelling in the temporal region, and abducens paralysis, is due to circumscribed meningitis at the tip of the petrous portion, does not hold good in all cases.

GOETERMANN (102, **Abducens paralysis after lumbar anæsthesia**) reports this accident after spinal injection of about one grain of tropacocain with adrenalin, coming on eight days after operation and lasting six weeks. The paralysis was nuclear and due to toxic absorption.

LACHMUND (103, **Study of the convergence reaction**) noted reflex pupillary paralysis in a girl affected with functional psychosis, in which the pupil remained oval and larger than its fellow during convergence on contraction.

LEVINSOHN (104, **Meiosis in reflex pupillary paralysis**) does not believe that this condition can be explained by lack of sensory stimuli or by sympathetic paralysis. The reaction after experimental irritation of the descending root of the sympathetic cannot be considered as analogous with Argyll-Robertson's pupil, as the former is followed by increased meiosis if light now enters the eye. As accommodation and accommodative contraction are not affected in reflex pupillary paralysis, the seat of the irritation must be in the sphincter centre. If this consists of two parts, one for light and one for accommodation reaction, disease of the first portion and irritation of the second would cause paralysis with meiosis. The latter is not spinal, but cerebral.

ABELSDORFF (105, **Unilateral reflex pupillary paralysis in oculomotor palsy**) reports a case of peripheral palsy, due to direct injury, which cleared up. The reflex paralysis is explained on the basis of associated motions.

HESS (107, **Physiology and pathology of the pupil**) tested the pupillo-motor reaction of various portions of the retina, using lights of equal intensity and size and also varying both these factors to produce isokinetic stimuli. The motor irritability of the retina diminishes more rapidly toward the temporal side. The motor area is comparatively small. In experimental pressure-blindness there was no reaction of the pupil. The outer members of the visual epithelium are the organs which take up the motor stimulus. It is not probable that there are separate visual and pupillary fibres,

but a single fibre probably conducts both impulses for some distance, while the centres are separate.

KRUSIUS (108, **Divergent strabismus in hypermetropia and the effect of mydriasis**) noted disappearance of a divergent squint after atropinization. In distant vision the paretic left ciliary muscle received a strong impulse which was communicated to the right eye as an associated inward motion. The squint again became manifest when the atropinized eye was corrected for distance or eserin was instilled.

LEVI (109, **Voluntary squint in primary position of fellow eye**) notes that in voluntary squint the motion is not unilateral, but that here too Hering's law of the simultaneous innervation applies. There is a progression from binocular vision overcoming dissociating prisms, and involuntary dissociation, to latent strabismus and to the voluntary control of unilateral squint.

BARTELS (110, **Simple phorometer for latent divergence**) uses a tangent scale and a Maddox rod at reading distance. Normal eyes show a divergence of 3 to 5 degrees, and there are constant oscillations probably due to varying accommodation and convergence.

OHM (111, **Retinal meridians after squint operation**) tested 10 cases of convergent, and 2 of divergent squint with Hirschberg's tropometer. Anomalous localization persists for some time after correcting operation. After convergence, crossed diplopia generally occurs. Later on there is an antagonism of normal and abnormal localization. Binocular triplopia and monocular diplopia were noted. Frequent change and alternation of fixing eye is advisable in this stage. Finally normal projection is established. Binocular vision is rarely perfect.

CASSIRER and LOESER (112, **Influence of rotation on nystagmus**) found in pathological nystagmus of neuropathics that horizontal nystagmus was abolished by turning toward the side of the nystagmus, but increased by turning in the opposite direction to the gaze. The pathological irritation of one labyrinth which causes primary nystagmus is overcompensated by the adequate irritation of the contralateral labyrinth due to rotation.

DROUIN (116, **Congenital amblyopia with divergent squint**)

was able to effect a complete cure in a woman of thirty-two by the use of Rémy's diploscope.

HEDON (117, **Post-operative oculomotor paralysis in otitis media**) explains a case of this sort associated with severe trigeminal neuralgia, particularly of the ophthalmic division, with conjunctival hyperæmia and lachrymation, as the result of an osteitis of the apex of the petrous portion.

LANDOLT (118, **Clinical results in muscle advancement**) reports a number of cases of advancement without tenotomy of the squinting muscle. In divergent squint he advances both interni.

WYLER (120, **Fleeting paralysis of ocular muscles**) saw a healthy subject aged twenty-nine years, with spontaneous diplopia due to paralysis of the superior oblique and internal rectus of the left eye. A cure was effected in three weeks by means of aspirin and galvanic current. The affection was an early symptom of multiple sclerosis.

As CULBERTSON (121, **Paralysis of ocular conjugate superduction**) remarks, conjugate paralysis of upward movements is a rare symptom and supposed to indicate a lesion in the quadrigeminal region. It is probable, however, that the symptom is not a direct one, for such a lesion as tumors destroying the corpora quadrigemina may leave these movements intact. Hence it is likely that the nuclei are affected only indirectly through pressure. ALLING.

Paralysis of extra-ocular muscles are not extremely rare in exophthalmic goitre. They affect only one eye usually and are due to lesion in the central nuclei. One of POSEY'S (122, **Ocular palsy in Graves' disease**) cases had palsy of the right superior rectus, the other had paresis of both external recti and of the left inferior rectus and inferior oblique. ALLING.

VEASEY'S (123, **Rhythmic alteration of palpebral fissure**) case was aged seven years, and had for some months been showing an upward motion of each upper lid, which occurred about twenty times a minute and was worse under nervous excitement. It was probably due to contraction of the levator as the frontalis was not involved nor were the pupils. Arsenic controlled the affection, which was a habit chorea. Posey has reported a similar case. ALLING.

ROENNE (124, **Disturbances of associated movements**) reports the case of a debilitated patient aged fifty with reflex pupillary paralysis, bulbar disturbance of speech, and bilateral paralysis of associated lateral motions, both voluntary and reflex, with perfect convergence and good vertical motility. Axes parallel, with occasional spasms of convergence. Post-mortem microscopic studies showed degeneration of the posterior longitudinal fibres with normal oculomotor nuclei. The loss of reflex abduction does not always mean destruction of the abducens nucleus.

STEPHENSON (125, **Associated movements of upper eyelid and jaw**) reports a case of synkinesis between the jaw and upper lid which was affected with ptosis. Patient aged nine had slight ptosis on the left side. When the lad chews, the right upper lid jerks rhythmically up and down. The same occurs when the mouth is opened and shut, or when the lower jaw is moved from side to side. Similar cases have been recorded by Fuchs, Frānkel, and Eales. These cases were discussed by Sinclair in the *Ophthalmic Review* for 1895, page 307. See also *Trans. Ophth. Society of London*, vol. iii., 1883, page 286, and xxiii., 1903, page 369.

The current explanation is that the levator palpebrarum, generally supplied by the third nerve, receives in these cases an additional supply from the fifth nerve, and so becomes associated with the muscles of mastication. Harman, however, explains "jaw winking" as an atavistic anomaly.

T. HARRISON BUTLER.

BOOK REVIEWS.

VII.—**A System of Ophthalmic Therapeutics.** Edited and chiefly written by Dr. CASEY A. WOOD, Chicago; illustrated and completely indexed; pages, 926. Chicago, Cleveland Press, 1909. Price, $6.00 net.

This exhaustive work, by the well-known author and judiciously chosen collaborators, undertakes to give in detail the non-operative treatment of eye diseases, including prophylaxis. The arrangement of the work is practical and logical in its sequence. After an historical introduction chapters follow on Classification of Ocular Remedies, Technique of Ophthalmic Therapy and Some of the Internal Remedies Used in Eye Diseases. Dr. W. Franklin Coleman contributes a chapter on Electricity in Ophthalmic Practice. Serum and Bacterial Therapy and Reactions in Diagnosis are treated by Dr. Ernest E. Irons. Dr. Frank Allport describes his scheme for the Systematic Examination of the Eyes of School Children, and the subject of Examination of Corporation Employees is treated by Dr. N. M. Black. Dr. Alfred C. Croftan has written on the Treatment of Systemic Diseases that Involve the Ocular Apparatus. Dr. D'Orsay Hecht treats in a similar way of Nervous Diseases. Dr. Frank Brawley gives the Non-Surgical Treatment of the Nose and the Accessory Cavities in their Relation to Eye Disease. A useful chapter on General Anæsthetics is furnished by Dr. H. D. Peterson. The remainder and greater part of the book is devoted to an alphabetical description of the Remedial Agents Employed in the Local Treatment of Eye Diseases, and to the Treatment of the Diseases of Each Part of the Eye. Ocular Hygiene, Errors of Refraction, and Non-Operative Treatment of Anomalies of the Orbital Muscles each have

a chapter assigned to them. Finally, Ocular Headache, Non-Operative Conduct of Ocular Injuries, and Preparation of the Patient and Surgeon for Ophthalmic Operations conclude the list of chapter headings.

The book contains an enormous amount of information, in fact much information which is usually inaccessible. It will be found to be of great convenience and of practical value to every ophthalmologist.

A. K.

VIII.—**Retinitis Pigmentosa.** By WILLIAM T. SHOEMAKER, M.D., Philadelphia. An essay which received the Alvarenga Prize of the College of Physicians of Philadelphia, July, 1908; 106 pages, with illustrations and colored plates. Philadelphia, J. B. Lippincott Co., 1908. Price $2.00.

This essay is based on the study of 17 cases occurring in deaf-mutes and a review of the literature. After general chapters on history and pathology, symptomatology, etiology, a complete analysis of the seventeen cases is given, with a summary of the data. A new feature of the investigation is a thorough examination of the blood and urine, which was made by Dr. John M. Swan in the Laboratory of Physiological Chemistry of the University of Pennsylvania; this examination unfortunately failed to throw any new light on the subject. Prognosis, treatment, and bibliography complete the volume. Though the author's conclusions do not bring anything particularly new, the essay is a painstaking and elaborate presentation of the subject, with an analysis of carefully examined cases. There are excellent water-color sketches of the fundus by Dr. Mary Buchanan, who has also plotted the visual fields.

A. K.

IX.—**Atlas and Epitome of External Diseases of the Eye.** By Professor Dr. O. HAAB, of Zurich. Edited, with additions, by GEORGE E. DESCHWEINITZ, M.D., Professor of Ophthalmology, University of Pennsylvania. Third revised edition. With 101 colored lithographic illustrations on 46 plates and 244 pages of text. Philadelphia and London: W. B. Saunders Company, 1909. Cloth, $3.00 net.

X.—**Atlas and Epitome of Ophthalmoscopy and Ophthalmoscopic Diagnosis.** By Professor Dr. O. HAAB, of Zurich.

Edited, with additions, by GEORGE E. DESCHWEINITZ, M.D., Professor of Ophthalmology, University of Pennsylvania. Second revised edition. With 152 colored lithographic illustrations and 94 pages of text. Philadelphia and London: W. B. Saunders Company, 1909. $3.00, net.

We are glad to welcome a new edition of Prof. Haab's well-known atlases. Their value needs no emphasis at this time. The revision and addition of new subject matter bring the books up to date, and will add to their popularity, especially as in this English form the reader enjoys the collaborative notes of the talented American editor.

A. K.